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THE IMPORTANCE OF EARLY AND ACCURATE DIAGNOSIS IN JOINT DISEASES.[1]

By NATHANIEL ALLISON, M.D. (Harvard),
Professor of Surgery, University of Chicago.

I HAVE taken for my subject the early and accurate diagnosis of joint disease because we have recently discovered that our own methods of diagnosis were not all that could be desired. I need not, I am sure, lay any emphasis upon the necessity for early diagnosis in joint disease nor stress especially the equally great necessity for accurate diagnosis, because without these two essential features to proper practice we add confusion and difficulty to a subject already sufficiently difficult and confused.

As you well know there is a tremendous literature on the subject of joint disease which is being added to extensively each year by books, pamphlets and lectures, almost all of which have for their purpose the recommendation of some new form of therapy for arthritis. Looking back through this mass of literature one is immediately struck with the rarity of any serious pathological studies of the lesions of joint disease. Indeed, the study of tissue changes is confined to a very few contributions which in consequence stand out as beacons, each contributor to the subject taking his bearings from them.

Due to a lack of accurate knowledge the nomenclature of the subject of joint disease has remained highly confused and inaccurate. A set of diagnostic terms is used which was invented before the day of the microscope, and which is based upon gross changes in the tissues, describing only the secondary and later changes which occur in the tissues as a result of arthritis.

It seems that what is most needed in the future study of arthritis is, in the first place, a clear knowledge of the tissue changes in the various types of the disease and in the second place, a nomenclature based upon these tissue changes. Not before we have secured these two things shall we be enabled to speak a common language, nor to understand our own efforts at therapy. After we have secured these and not until then will reported observations on arthritis have the ring of scientific truth.

Perhaps many of the difficulties which arise in a study of arthritis, may be reduced, provided the essential tissues of the joint structure are chiefly considered. Bone is not so difficult to understand if it is thought of as a tissue rather than a solid substance, a living tissue which in fact houses within itself the marrow. The marrow is without doubt the important tissue in so far as disease is concerned. Think then of the marrow when you think of bone in its reactions to disease. Beyond the marrow changes all the reactions of bone to a disease process are secondary. Atrophy and hypertrophy of bone are secondary changes. Ely has said with truth that disease is not of the bone, but disease is in the bone.

Perhaps also when considering the involvement of a joint by disease the process is more easily visualized if we consider the synovial membrane as the essential structure. All disease processes of the joints attack the synovial membrane primarily. Other changes are secondary. There is one exception to this rule. In degenerative arthritis the primary change is in the articular cartilage.

For our own use I believe it is reasonable to approach diagnosis and classification of joint diseases along these lines:

Disease processes attack the synovial membrane and the marrow in the bone. Secondary changes rapidly follow. The close association anatomically and clinically which exists between the marrow and the synovial membrane, is of great significance, meaning that when one is involved by a disease process, almost immediately the other will be involved. This fact should never be lost sight of, because it means practically that arthritis is a disease process which involves the articular cavity as well as the marrow in the ends of the bones. Albert Key has shown how quickly particles of carbon introduced into a joint cavity find their way into the marrow spaces of the bone.

In one type of chronic arthritis, so-called degenerative arthritis, the primary change is in the articular cartilage. The synovial membrane and marrow are not to any marked degree involved. An appreciation of these simple facts will aid us materially both in early diagnosis and in the use of better diagnostic terms in joint disease.

About six years ago we found in my service at the Massachusetts General Hospital that we were not making sufficiently early or accurate diagnosis in the instances of arthritis which were passing through the wards of the hospital. As shown by my associate, R. K. Ghormley, in forty-one cases which were held by clinical tests to be due to tuberculosis, twenty-seven were proven to be due to tuberculosis by tissue study and guinea-pig test. In 66% the diagnosis was correct. In fifty-two cases which were held to be of probable tuberculous origin, after clinical examination twenty-seven were proved to be due to tuberculosis and the diagnosis was 52% correct. But more important still were forty-two cases of arthritis in which tuberculosis as a possible cause was not considered clinically, but of which twenty-three were proven to be due to tuberculosis by tissue examination and guinea-pig test. In 55% the diagnosis was correct. If this range of error exists in the case of tuberculosis, it seems fair to conclude that an even greater margin of error exists in other types of joint involvement.

In consequence of this discovery we have had established a routine for diagnosis in all cases of arthritis which have passed through the service. This routine includes the following: (i) All clinical data, (ii) readable X ray photographs, (iii) serologic tests, (iv) skin reactions, (v) chemical and bacteriological examination of the joint fluid, if obtainable, (vi) smear stain of the joint fluid, if

[1] Read at a meeting of the Post-Graduate Work Committee of the New South Wales Branch of the British Medical Association on January 22, 1930.

obtainable, (vii) guinea-pig test of the joint fluid, if obtainable, (viii) Wassermann or Kahn test on the blood and joint fluid, if obtainable, (ix) the removal of tissue from the joint for microscopic study, (x) guinea-pig test on tissue removed from the joint, (xi) bacteriological study of tissue removed from the joint, (xii) careful recording of all data, including photomicrographs of sections and reduction of X ray photographs. The material thus collected is carefully filed and kept for reference.

We have secured in the past six years complete records on 289 cases of arthritis studied in this manner, all of which will be the basis for publication in the near future. Before I try to put before you the value of such methods of study in a hospital service, allow me to refer but briefly to our difficulties in classification.

The literature of arthritis shows clearly how hard a thing it is to throw off old habits in nomenclature, no matter how bad these habits may be. In 1909 Nichols and Richardson published the results of a careful study of tissue changes in arthritis. They found from this study that two types of disease practically covered the range of tissue change, proliferative arthritis and degenerative arthritis. The one was primarily a change in the synovial membrane and marrow, while the other was a primary change in the articular cartilage. These valuable observations have not passed unnoticed; each writer on arthritis acknowledges their truth, but nevertheless sticks to older terms for fear he will not be understood. Knaggs, for instance, writing a book on bone disease last year still uses osteo-arthritis and rheumatoid as two of his types. Timbrell Fisher also writing a book on arthritis last year tries to advance by speaking of a type one and type two arthritis. In this he followed the example of Ely, but unfortunately Ely's type one is Fisher's type two and *vice versa*. Pemberton writing in 1929 a book on arthritis and rheumatoid conditions has tried to straddle the difficulty by using compound names, as, for instance, atrophic or proliferative arthritis, hypertrophic or degenerative arthritis. In this he realizes his position and blames it upon an American committee on rheumatism.

Of all writers on this subject Ely has kept the clearest head, recognizing fully the importance of Nichols's and Richardson's observations. Ely's trouble is that he has recommended the use of the terms type one and type two arthritis which is confusing to many to remember which is which. Nichols and Richardson themselves used the term *arthritis deformans*, a term invented by Virchow and one which means nothing at all beyond what long clinical usage has brought to it. The same is true of the terms osteo-arthritis, rheumatoid arthritis, atrophic arthritis and hypertrophic arthritis. All these terms do not describe primary changes in the tissues, but rather describe later secondary changes.

It seems that well established clinical terms are exceedingly hard to displace, no matter how erroneous or confusing they may be.

For our own purposes of study we have grouped our instances of arthritis under several heads, using terms which give their exact ætiological factor or which point out the primary tissue change.

We believe that this constitutes a true nomenclature based upon sound pathology, as follows: (i) traumatic arthritis, (ii) proliferative arthritis of known bacterial origin, (iii) proliferative arthritis of uncertain origin, (iv) degenerative arthritis, (v) arthropathies, syringo-myelia, *tabes dorsalis*, (vi) constitutional dyscrasies, gout, hæmophilia, hysteria.

Of traumatic arthritis and arthritis of known bacterial origin I will speak but little; diagnosis is not difficult, but should be made with accuracy and without delay. This is certainly true of the types of joint involvement which have the pus-producing organisms as their cause. Tuberculosis demands a special word. Diagnosis is very important because both the child with a tuberculous joint and the child suspected demand positive proof of the nature of the disease. This is obvious. Our attempts at therapy may be harmful otherwise. Knowing the actual cause gives us ample reason for treatment, either operative or conservative. Not knowing the cause may result in disaster.

After our experience of the past six years we feel that biopsy, the removal of tissue from a joint for examination under the microscope and guinea-pig inoculation, is a proper diagnostic procedure, entirely justifiable from the patient's point of view.

Of proliferative arthritis of uncertain origin I feel that we have added by our study an observation of great importance. This is the type of arthritis which is multiple, which affects younger people, and which goes on to ankylosis. The synovial membrane and marrow are involved primarily. Secondarily, the joint cartilages are destroyed by granulations and pannus. Sections of synovial membrane and bone show a characteristic picture, a picture under the microscope which is as clear cut and valuable as is the discovery of tubercle bacilli in tuberculous tissue. There are scattered about in the marrow and synovial membrane focal collections of small round cells, lymphocytes, which are not seen in any other type of arthritis. This characteristic of proliferative arthritis is mentioned by Ely, by Timbrell Fisher and perhaps others, but no importance has been attached to it. We feel that it is absolutely diagnostic of this type of disease and that it is the primary change in the tissue. The joint tissues in proliferative arthritis are not the seat of bacterial invasion. This tissue response is probably due to toxins. Repeated efforts to grow by culture bacteria from the freshly removed synovial membrane have resulted in our own hands and those of Hans Zerisser in nothing. Zerisser is of the belief that this type of arthritis is an allergic state and in this view we are in agreement. R. V. Graham's work with cultures and vaccines made on the whole blood of the patient adds an interesting angle to this belief. I have not time to discuss all the phases of this long neglected type of arthritis. Early and accurate diagnosis is what is demanded.

Degenerative arthritis along with the arthropathies and the constitutional disturbances which have joint phenomena, are not difficult subjects to understand.

We have found that therapy is much aided by early and accurate diagnosis.

Methods of joint examination and study can be well carried out in any hospital or office, so that we reach a higher percentage of correct diagnosis and with that a better knowledge of what we are doing for our patients. In conclusion, let me urge the necessity for better methods of diagnosis and classification in arthritis, believing that more satisfactory results will thereby be secured.

A SHORT HISTORY OF SCURVY IN AUSTRALIA.

By J. Burton Cleland, M.D.,
Marks Professor of Pathology, University of Adelaide.

The recent outbreak of scurvy amongst the natives at Hermannsburg Mission Station in Central Australia has drawn attention to this disease, at one time so important. The public in South Australia showed much sympathy with the aborigines and fruit growers very generously contributed lemons and oranges as antiscorbutics.

The disease scurvy has a particular interest for us as Australians, inasmuch as the measures taken against it by Captain James Cook were entirely successful and by this success he was enabled to make those great additions to geographical knowledge that fell to his lot. Admiral Wharton,[1] the editor of his journal, has expressed the opinion that Captain Cook's "greatest triumph was not so much along the lines of geographical discovery as in his success in overcoming the scurvy. How injurious the scurvy might be is well illustrated in the case of Lord Anson's[2] 'Voyage Round the World'." It appears that scurvy overtook the expedition soon after passing Cape Horn. The very graphic description given in the account of the voyage is so full of medical detail that it is generally believed it was compiled by a professional man. The account is worth repeating here. The date was 1741.

> Soon after our passing Straits Le Maire, the scurvy began to make its appearance amongst us . . . This disease so frequently attending all long voyages, and so particularly destructive to us, is surely the most singular and unaccountable of any that affects the human body. For its symptoms are inconstant and innumerable, and its progress and effects extremely irregular; for scarcely any two persons have the same complaints, and where there has been found some conformity in the symptoms the order of their appearance has been totally different. However, though it frequently puts on the form of many other diseases, and is therefore not to be described by any exclusive and infallible criterions; yet there are some symptoms which are more general than the rest, and, therefore, occurring the oftenest, deserve a more particular enumeration. These common appearances are large discolored spots dispersed over the whole surface of the body, swelled legs, putrid gums, and above all, an extraordinary lassitude of the whole body, especially after any exercise however inconsiderable; and this lassitude at last degenerates into a proneness to swoon on the least exertion of strength, or even on the least motion. This disease is likewise usually attended with a strange dejection of the spirits, and with shiverings, tremblings, and a disposition to be seized with the most dreadful terrors on the slightest accident. Indeed it was most remarkable, in all our reiterated experiences of this malady, that whatever discouraged our people, or at any time damped their hopes, never failed to add new vigour to the distemper; for it usually killed those who were in the last stage of it, and confined those to their hammocks who were before capable of some kind of duty; so that it seemed as if alacrity of mind and sanguine thoughts were no contemptible preservatives from its fatal malignity.
>
> But it is not easy to complete the long roll of the various concomitants of this disease; for it often produced putrid fevers, pleurisies, the jaundice, and violent rheumatic pains, and sometimes it occasioned an obstinate costiveness, which was generally attended with a difficulty of breathing; and this was esteemed the most deadly of all the scorbutic symptoms. At other times the whole body, but more especially the legs, were subject to ulcers of the worst kind, attended with rotten bones, and such a luxuriance of fungus flesh, as yielded to no remedy. But a most extraordinary circumstance, and what would be scarcely credible upon any single evidence, is, that the scars of wounds which had been for many years healed, were forced open again by this virulent distemper. Of this there was a remarkable instance in one of the invalids on board the *Centurion*, who had been wounded above fifty years before at the battle of the Boyne; for though he was cured soon after, and had continued well for a great number of years past, yet, on his being attacked by the scurvy, his wounds, in the progress of his disease, broke out afresh, and appeared as if they had never been healed. Nay, what is still more astonishing, the callus of a broken bone, which had been completely formed for a long time, was found to be hereby dissolved and the fracture seemed as if it had never been consolidated. Indeed, the effects of this disease were in almost every instance wonderful; for many of our people, though confined to their hammocks, appeared to have no inconsiderable share of health, for they ate and drank heartily, were cheerful, and talked with much seeming vigour and with a loud, strong tone of voice; and yet on their being the least moved, though it was only from one part of the ship to another, and that in their hammocks, they have immediately expired; and others who have confided in their seeming strength, and have resolved to get out of their hammocks, have died before they could well reach the deck; and it was no uncommon thing for those who were able to walk the deck, and to do some kind of duty, to drop down dead in an instant, on any endeavours to act with their utmost vigour, many of our people having perished in this manner during the course of this voyage.

There are, I think, few more graphic accounts of scurvy in the English language.

As regards the occurrence of scurvy actually in Australia, I have already given a short epitome.[3] This will be briefly summarized here and further records will be given.

It seems that scurvy appeared at Sydney soon after the arrival of the first fleet. The *Supply* was sent to Lord Howe Island to endeavour to procure turtle in the hopes of checking it. The transports *Surprise, Neptune* and *Scarborough* arrived in 1790 with many patients suffering from scurvy and dysentery on board. On the *Neptune* many died in irons and often the death would be concealed by those next to the deceased for the purpose of obtaining the dead man's allowance of provisions, chance or the offensiveness of the corpse finally attracting attention to the death. Several other transports, up to the year 1801, likewise arrived with scurvy on board. Among exploring expeditions it was found from time to time that scurvy attacked some

of the members. When Baudin arrived at Port Jackson on June 22, 1802, he had only twelve men able to work the ship on account of the ravages of scurvy. Captain Flinders contracted scorbutic ulcers on his legs during his voyage around Australia in 1803. The scurvy attacked members of Captain King's expedition in 1819, Oxley's men suffered in 1823, Major Mitchell's in 1832 and Major O'Halloran's expedition up the Murray in 1841 and in Sturt's explorations in Central Australia in 1845 Mr. Poole died of the disease and the leader was himself attacked. Similarly scurvy occurred in A. C. Gregory's party, in Stuart's party and on the expedition of Burke and Wills. Dr. S. D. Bird considered that the indescribable leg-bound feeling, the shortness of breath, the sensation of pain and helplessness and the clearness of vision to the last clearly indicate that Burke and Wills suffered from scurvy. One of Major Warburton's camel men suffered from it in 1873. In 1894 an editorial appeared in the *Australasian Medical Gazette* on bush scurvy; a number of attacks had recently occurred. In 1901 W. Macansh described the condition in a boundary rider from the Riverina.

The following references were not included in my former summary of the occurrences of this disease in Australia epitomized above; they are given in somewhat greater detail.

Collins,[4] under date April, 1796, mentions that the Americans as a preventive against scurvy made frequent use of acids in the diet of their seamen and of fumigations of tobacco in their between-decks.

Dr. W. L. Crowther,[5] of Hobart, has given references to the occurrence of scurvy. In July, 1804, the *Ocean* after a tempestuous voyage of thirty-three days from Port Phillip, brought a large sick list, mostly of scorbutic patients. The Lieutenant-Governor Colonel Collins who had arrived in the previous February, had also men with scurvy among those who accompanied him. Scurvy, diarrhœa and catarrh proved to be the prevailing diseases in the infant years of the settlement. Between August 9 and November 9, 1804, of forty-seven deaths scurvy accounted for thirty. The sourness of the ground rendered it difficult for them to grow vegetables and recourse was had to kangaroo meat as an antiscorbutic.

James Backhouse[6] has several references to scurvy. He mentions its presence amongst the prisoners at Macquarie Harbour in 1832. In 1833 he notes that scurvy had increased amongst the prisoners at Port Arthur, though their health generally was good. At the penal settlement on Tasman's Peninsula in November, 1834, he states that "the prisoners living on salt meat and being defectively supplied with fresh vegetables, a large number were suffering from scurvy." Amongst the ironed gangs on the Blue Mountains in 1835 he notes that many of the prisoners became affected with scurvy.

In the Governor's dispatches in the Mitchell Library[7] in Sydney, notes of which have been made by Dr. F. Watson who kindly enabled me to peruse them, there is a reference to scurvy in letters from Governor Gipps to Lord Glenelg in 1838. It appears that 114 men suffering from scurvy had been transferred to hospital from the convict transport *Lord Lynedoch*. The disease had appeared just after leaving the Cape of Good Hope; one hundred and fifty patients had been admitted and fifteen had died.

In a report by the Colonial Surgeon of South Australia quoted by Dutton,[8] it was stated that most of the cases of illness that had so far occurred in this State were of a scorbutic nature. This report would bear a date before 1846.

Cenek Paclt,[9] a Czech traveller in Australia from 1856 to 1862, was attacked with scurvy in Queensland. William White,[10] the ornithologist, uncle of Captain S. A. White, of South Australia, was attacked by scurvy at Cleveland Bay (now Townsville) in 1867. Maiden[11] stated that at the fourth monthly meeting of the Royal Society of New South Wales in 1867 Edward Bedford contributed a paper on the reappearance of scurvy in the merchant service. Apparently Maiden's is the only published record of this paper.

Giles[12] states that in August, 1871, Mr. Ross, leader of the exploring expedition in connexion with the construction of the overland telegraph line, suffered severely from scurvy at the Northern Territory end of the line. Amongst the parties in 1883 and 1885 travelling cattle overland from Queensland to Kimberley in Western Australia, a journey which took two years and eight months, scurvy was frequent.[13]

W. A. T. Lind[14] describes twelve cases occurring amongst the inmates of the Idiot Cottages at Kew, Victoria; four of the patients died and in most of the patients limbs or joints were swollen. F. W. Litchfield[15] stated that infantile scurvy was uncommon in Australia and that he saw only one or two patients a year. C. Madden,[16] of Sydney, described a case of scurvy in a child aged nine and a half years, who would eat only bread, butter and sugar and occasionally prawns, oysters or fish. There was pain on pressure near the joints and some swelling, softness and tenderness of the gums. Rapid improvement occurred under treatment. D. W. Neville[17] described the case of an old-age pensioner of sixty-eight who had lived on bread and cheese with some butter and occasional jam. He had swollen, spongy gums and severe anæmia. He rapidly improved on proper diet. E. W. Frecker[18] described a case in a man aged fifty, who did not eat vegetables and fruit. J. W. Grieve,[19] before the Melbourne Pædiatric Society, gave notes on a baby girl of nine months suffering from scurvy. H. D. Stephens,[20] of Melbourne, described a case in a boy of ten months.

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(1) W. J. L. Wharton: "Captain Cook's Journal," 1893, page L.
(2) George Anson: "A Voyage Round the World in the Years MDCCXL, I, II, III, IV."

[3] J. B. Cleland: "Contributions to the History of Diseases in Man in Australia: (*d*) Scurvy," *Sixth Report of the Microbiological Laboratory*, Report of the Director-General of Public Health, 1915, New South Wales, page 273.
[4] David Collins: "An Account of the English Colony in New South Wales," edited by James Collier, page 276.
[5] W. L. Crowther: "Some Medical Aspects of the Settlements on the River Derwent, 1803-1805," THE MEDICAL JOURNAL OF AUSTRALIA, March 12, 1927, page 370.
[6] James Backhouse: "A Narrative of a Visit to the Australian Colonies," 1843.
[7] Governor's Dispatches, 16, Gipps to Glenelg, No. 125, August 17, 1838, and No. 148, September 24, 1838, Mitchell Library.
[8] F. Dutton: "South Australia and Its Mines, with an Historical Sketch of the Colony," 1846, page 102.
[9] J. V. Danes: *Royal Australian Historical Society Journal and Proceedings*, Volume VIII, Part 4, 1922, page 212.
[10] S. A. White: "Life of Samuel White," page 20.
[11] J. H. Maiden: "History of the Royal Society of New South Wales," *Journal and Proceedings of the Royal Society of New South Wales*, 1918, page 316.
[12] Alfred Giles: "Exploring in the Seventies," *The Register*, page 103.
[13] "The Inlander," Volume 6, 1920, page 10.
[14] W. A. T. Lind: "Some Interesting Details of an Outbreak of Scorbutus," THE MEDICAL JOURNAL OF AUSTRALIA, August 9, 1919, page 107.
[15] THE MEDICAL JOURNAL OF AUSTRALIA, November 6, 1920, page 446 (Report of a Meeting of the New South Wales Branch of the British Medical Association).
[16] C. Madden: "A Case of Scurvy Occurring in Melbourne," THE MEDICAL JOURNAL OF AUSTRALIA, December 1, 1923, page 571.
[17] D. W. Neville: "A Case of Scurvy," THE MEDICAL JOURNAL OF AUSTRALIA, January 19, 1924, page 63.
[18] E. W. Frecker: "A Case of Scurvy," THE MEDICAL JOURNAL OF AUSTRALIA, February 10, 1927, page 274.
[19] THE MEDICAL JOURNAL OF AUSTRALIA, July 16, 1927, page 102 (Report of a Meeting of the Melbourne Pædiatric Society).
[20] THE MEDICAL JOURNAL OF AUSTRALIA, March 9, 1929, page 330 (Report of a Meeting of the Melbourne Pædiatric Society).

AN OUTBREAK OF SCURVY WITH JOINT LESIONS IN AUSTRALIAN ABORIGINES IN CENTRAL AUSTRALIA.

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AND
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IN August, 1929, a short anthropological expedition, under the auspices of the Board of Anthropological Research of the University of Adelaide and partly financed by the Rockefeller Foundation Fund administered by the Australian National Research Council, was made to the Hermannsburg Mission Station on the Finke River, about eighty-five miles west of Alice Springs. A large number of aborigines, over 320, were congregated here, partly owing to the five years' drought in the district and partly to the removal of natives from the neighbourhood of Alice Springs on account of the building of the railway to Stuart. On arrival it was found that a number of natives were sick, their illness having been diagnosed as beri beri, and that forty had died during the year. The majority of these had had swollen or sore mouths and some had had swollen joints as well. Many of them had died apparently from superadded complaints, such as lung trouble.

On examining the sick, it was found that all had ulcerated gums (marginal gingivitis), often with foul breath and that in many the gums were bleeding and swollen and often their spongy summits nearly reached the level of the crowns. The gums were red, sometimes a dusky red. We were informed that the teeth sometimes came out and had been found lying on the bedding beside patients. The roots of some teeth were partly exposed. The natives' teeth, it may be mentioned, are normally remarkably sound, large and clean. In addition a number of the patients had had swollen knees, and one had had an affected shoulder and another a swollen elbow. Some of these patients had been ill for two or more months. The swellings of the joints or their neighbourhood had presumably been due to extravasations of blood. In some cases fluid still appeared to be present. In others the knee joints were now flexed and the patients seemed unable to relax them. The hamstring muscles were in some patients tense and contracted. Attempts at extending the legs caused much pain and were almost entirely unsuccessful. Probably organization of the extravasated blood had led to adhesions.

A diagnosis of scurvy with a special tendency to hæmorrhages into the knee joints or near them was made. The available scanty supply of antiscorbutics was generously given up by the staff for the use of the natives. These consisted of oranges, turnips and some cabbage leaves and turnip leaves. No lemons were available.

On inquiry it was found that the number of natives on the station had been 383 in January, varying down to 332 in May. Of these in June 231 were natives from the mission station and 101 from Alice Springs. The mission blacks comprised able-bodied, old and infirm and children. As owing to the prolonged drought there were practically no natural foodstuffs (animals, including kangaroos and rabbits, lizards *et cetera* or seeds and roots) available in the neighbourhood of water, practically the whole population concerned received their food supply through the mission. Between January 28 and April 12 by Government order no firearms were allowed to be in the possession of natives, this following the killing of a white man, Henty, 250 miles north-east of Hermannsburg. This cut off the supply of occasional wallabies and kangaroos. The mission books show that in May 8,966 pounds of white flour were issued to or bought by the natives, 1,000 pounds of sugar, 244 pounds of polished rice, 220 pounds of pearl barley, 302 pounds of split peas, 226 pounds of dried fruit (chiefly apricots), forty pounds of tallow, 160 pounds of jam, 36 tins of condensed milk, saccharine for sweetening purposes, tea, a little tobacco and some coffee. In June these figures were mostly rather increased (for example, 10,341 pounds of flour, 840 pounds of rice, 360 pounds of pearl barley and 400 pounds of split

peas). Between January and March dried peas and dried fruit were not supplied. In January five head of cattle and seven sheep were killed, in February two and six respectively, in March none and eleven, in April two goats, in May two head of cattle and in June two head of cattle. Fourteen tins of meat were sold in April.

It thus appears that these natives received a little more than one pound of white flour per head per day and about three pounds of sugar, one to three pounds of rice and a little more than one pound each of pearl barley and split peas per month. A little mutton fat was also issued. The meat was in amount equivalent to two bullocks (or bulls) or less per month recently. The flour was mostly given as flour soup with in the later months the dried fruit added and sweetened with saccharine. The sugar was taken in the tea. These foods were supplemented to some small extent by a few euros (*Macropus robustus*), rock wallabies (*Petrogale lateralis*) and lizards, no seeds, but a few yelka (lily) bulbs obtained by the more able-bodied.

The following table shows the average daily ration supplied to the natives with the respective food values, in regard to protein, carbohydrate and fat, calculated in grammes.

Food.	Amount in Pounds.	Protein in Grammes.	Carbohydrate in Grammes.	Fat in Grammes.
Flour	1·0	49	340	5
Sugar	0·1	0	42	0
Rice	0·066	1·5	13	0
Barley ..	0·033	1	7	0
Split peas ..	0·033	3	8	1
Meat	0·1	9	0	1
TOTAL		63·5	410	7
Total allowing for 10% loss in cooking, digestion, etc.		57	369	6

Caloric value of assimilated food 1785

This meagre dietary supported life efficiently, as the natives were not emaciated, were mostly in excellent general condition and were able to carry out their normal activities unless actually ill.

The outstanding feature of the ration is its gross deficiency in all accessory food factors. Xerophthalmia, rickets, beri beri and scurvy should all be represented in view of this lack of vitamins. Scurvy appeared to be the only malady that actually appeared. Xerophthalmia may have been a factor in some of the many eye conditions, but trachoma and corneal injuries were sufficient explanations of these in so far as we were able to judge. Rickets was certainly not present. Beri beri was suggested by the tenderness of the calves of many of the patients, but the knee jerks were active except in a few of the patients with hæmorrhage into the knee joint. Swelling of the legs was present in two patients only. Further, no areas of anæsthesia were present and no asthenia apart from general debility or joint effusion.

In contrast to the general good condition of the natives at Hermannsburg, a family of bush natives who came in from the west, were very thin and emaciated, a poor infant at the breast having a wizened, famished face.

An interesting feature was the prevalence of swellings in or around the knee joints. This was the lesion that chiefly incapacitated the patients and was the condition that often indefinitely prolonged convalescence. We attributed the condition to hæmorrhages into the joint or into the muscles and tissues around. Its prevalence we thought might be due to the habit of the natives of sitting with their knees acutely bent under them. This seems their invariable habit and must surely place considerable strain on the structures round the knee joint. Then, when scurvy had developed, these tissues subjected to strain would be especially likely to suffer from hæmorrhages.

Details of Cases.

CASE I. Richard whose father was a pure-blood aboriginal and whose mother was a half-caste aboriginal and Syrian, aged eighteen, had been three months ill with swellings of his knees and ankles and at one time intense "expectoration" (probably salivation). Since his illness he had had split peas, white rice and dripping with sugar, dried plums and apricots and lime juice as food and drink without obvious improvement. His condition had been diagnosed as beri beri and rice had been ordered, but the kind supplied was polished. The patient was lying in bed, weak and emaciated, with his legs drawn up on one side and acutely flexed and he dreaded any attempt at touching and especially moving the legs. A general examination revealed a very definite coupled-beat pulse, suggesting a dicrotic pulse, of 108. There was a heaving impulse over the præcordial area, the apex beat was not recognizable. The cardiac dulness was normal and the sounds clear. The lungs were clear, the abdomen scaphoid, the liver and spleen not enlarged. An examination of the legs revealed some indefinite swelling of the flexed knees, the hamstring muscles tensely contracted, apparent wasting of the muscles of the thighs and legs (the lower limbs of these natives are naturally thin), both knee jerks slightly increased with the contraction extending to the flexors of the thigh and calf muscles, slight ankle clonus on the right and definite on the left, plantar reflex flexor on the right and slighter on the left. The Gordon reflex was present, the left calf was very tender, no tenderness was present over the sciatic nerve, there was no anæsthesia and no œdema of the ankles was found. The hand grips were good, the ordinary arm reflexes were present and the tendon reflexes were not exaggerated, but the direct muscle excitability was exaggerated. On examining the mouth there was a fœtid smell and an acute ulcerative marginal gingivitis, but without obvious swelling of the gums. The mother said that the mouth had been greatly swollen during the illness. With one orange a day the patient became much brighter. Nevertheless he developed considerable œdema of the left ankle. This had decreased a little before we left, but he complained of pain near the sciatic notch. Nothing could be detected here. When the expedition left twelve days after its arrival, this patient's pulse was regular and good. Shortly after our departure he rapidly went down-hill and died.

CASE II. Cora, sister of patient I, aged three, had been ill for a short while, complaining of pains in the knees and not sleeping therefrom. She was sitting with the knees acutely bent. The knees could be straightened, but this caused much pain. There was no swelling or œdema of the knees or ankles. The right knee jerk was brisk, the left not obtained. Both ankle jerks were brisk and without clonus. The plantar reflexes were not obtained. The pulse was 108 and irregular. The lungs were clear

and the abdomen normal. The gums were spongy and hæmorrhagic and partly covering the crowns of the teeth. Rapid improvement took place when oranges were supplied.

CASE III. Cornelius, brother of patients I and II, aged six, had a slight convergent squint with a right lenticular opacity. The pulse was 108 and irregular. The child was sitting about. There was tenderness of the left calf, but no tenderness of the bones. The knee jerks were active, the ankle jerks very brisk and without clonus. The face was swollen and the gums bleeding, swollen and partly covering the crowns of the teeth. This child rapidly improved under treatment.

CASE IV. Ruth was mother of patients I, II and III. She complained of weakness. Both calves, especially the left, were tender. The knee jerks were present, the ankle jerks very brisk, the plantar reflexes flexor. The pulse was 88 and regular. The gums were ulcerated. She rapidly improved. Another son, aged about seven, had died from scurvy four days previously and another daughter, aged about two, only two or three weeks before.

CASE V. Bernhard was a pure-blood aboriginal, aged about thirteen. He had been ill for four months, having diarrhœa at times, occasionally with blood. He was lying in bed on his back with the knees drawn up and whimpering with pain if touched. There was definite swelling of the suprapatellar pouch of the left leg which was very tender, but not hot. The legs generally were tender and especially the calves, the ankle jerks were present and the plantar reflexes flexor in type. There was free movement of the arms with no tenderness. The heart beat was diffuse, propulsive in the second and third spaces, indrawn in the fourth, the pulse 120 and regular. The abdomen was scaphoid. The gums, especially the upper, were spongy on the lingual surface, the tongue white and smooth with lateral dark pigment patches (a common racial characteristic).

One evening about three days after our arrival, the news came that Bernhard had suddenly died. Running over to the hut, we found that this was not true. The boy seemed *in extremis*, the mother weeping bitterly and incidentally spitting promiscuously, the father kneeling beside him. The pulse as we arrived was just returning and the child seemed to be suffering from pain in the region of the heart which seemed to come in paroxysms and catch him. Soon the boy was obviously improving. Next day he seemed much better, enjoying the fruit. He remained in this improved condition for some days, but then became worse again, in spite of receiving some oranges. The parents were rather troublesome and refused to obey instructions and the family had to be returned to the main camp and so the boy could not be retained in the "hospital" under closer observation. The treatment advocated may thus have lapsed in part, but the child received at least some oranges. A fortnight after our arrival he was going down-hill. The day before our departure we found him semicomatose with mucus in his throat, a fair pulse of 120, much œdema of the left ankle and up the tibia and an early bedsore over one trochanter. Death occurred about half an hour later. Our utmost endeavours failed to get permission for an autopsy.

CASE VI. George was a pure-blood aboriginal, about twenty-five years old. He was lying in bed curled up and unable to extend his legs. The left knee was slightly swollen and resisted movement. There was no tenderness of the arms. The pulse was 104 and regular. The gums were slightly swollen, the tongue pale with lateral patches of pigment. He quickly improved in general condition, though his legs remained much the same.

CASE VII. Sam was a pure-blood aboriginal, aged about fifty-five (he looked much older). He had been in bed for five months and was lying on his back with the legs semiflexed and everted. The left knee was very swollen, the left calf hard and infiltrated, the left tibia swollen, but there was no tenseness of the hamstring muscles. Both ankles were œdematous. The ankle jerks were present. No cardiac dulness was detected, the apex beat was not recognizable, the pulse 84, regular, but only just recognizable. The gums were red and spongy, especially on the lingual aspect and there was a foul smell. This patient whom we looked on as quite an old man, made no real improvement and died shortly after our departure.

CASE VIII. Maria, a pure-blood aboriginal, aged about fifty-five, had a fallen-in nose and perforated palate (? gangosa, probably not syphilitic). The gums were red with slight ulceration.

CASE IX. Doris was a half-caste, aged thirty. She had been ill for six months. She was lying on her side with the legs bent up and whimpered when touched. The knees were slightly swollen and flexed and she complained of pain on any attempt to extend them. There was slight tenderness of the legs. The arms were normal. The pulse was 110, regular, but weak and of low tension. The apex beat was prominent. The gums were slightly spongy, bleeding and ulcerated. Under treatment she rapidly became more cheerful and when we left was gradually extending the legs in which contraction of the muscles seemed the chief trouble.

CASE X. Abel was a pure-blood aboriginal, aged about forty-six. He was sitting and lying comfortably in bed, having been ill for about four months. He had improved and walked about and then relapsed. He now had a greatly swollen right knee containing fluid. It was slightly warmer than normal and slightly tender. The left elbow had been swollen and stiff for about three days, the swelling being rather diffuse and only slightly tender. The gums were ulcerated and slightly spongy. He quickly improved generally, the left elbow soon becoming normal.

CASE XI. Amanda was a pure-blood aboriginal, aged about twenty-six. She was sitting in a crouched position and whimpering. Both knees were very tender and rather swollen, as also was the right shoulder. The pulse was 120. There were slight sponginess and redness of the gums, especially on the lingual aspect. A marked improvement occurred under treatment in a few days.

CASE XII. Emma was a pure-blood aboriginal, aged eighteen. The knees were considerably swollen, especially the right, where there was tenseness in the popliteal space. The gums were swollen, red and ulcerated on both labial and lingual aspects. On the palatal aspect of the right molars the gums were nearly level with the crowns. This girl soon improved.

CASE XIII. Ningara was a pure-blood aboriginal, aged sixty to seventy. Her front teeth only were present. The gums were spongy and extensively ulcerated.

CASE XIV. Matilda was a pure-blood aboriginal, aged about fifty-five. She was weak, but the knees were not tender. The gums were swollen and ulcerated with in places some exposure of the roots.

In addition there was a number with mild and incipient scurvy that readily and rapidly became normal when orange juice was available.

THE EARLIER STAGES OF RENAL DEGENERATION.

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THIS is intended to be a practical discussion. So much has been written on renal degeneration and renal function that I can scarcely hope to state anything new. What I have endeavoured to do is to present a few facts in such a way that the reader will readily grasp their practical significance and feel that the problem of detecting and dealing with the early stages of renal degeneration is at least clarified if still not made exceedingly easy. It is from that angle that I have approached the subject; having reviewed such writings as have been

available, I feel that there may be some who will be glad to consider the subject in the light in which it appears to me.

I want to preface the discussion with a reference to two conceptions which are sometimes in danger of being misconstrued. We regard albuminuria as a frequent but not invariable sign of renal disease; but some of us, I fear, do not fully appreciate the fact that even when renal degeneration has progressed a considerable distance, we may obtain specimens of urine in which not even a little albumin can be demonstrated. There is an abundance of evidence to prove this fact. And in the matter of urinary specific gravity we learn soon enough that there is a fairly wide normal range even in one individual, depending somewhat on eating and drinking and climatic conditions and there is a danger that we too greatly discount all specific gravity readings and so discard one of the most valuable indications of early renal degeneration. I shall refer to this point again.

The function of the kidneys is to remove the urinary constituents from the blood, so that the composition of the blood may remain constant. Hence renal efficiency is the measure of success with which this function is performed. The elimination of toxins or bacteria not normally present in the blood stream is a secondary renal function.

Of greatest importance to the practitioner is some reliable method of determining the condition of the kidneys when signs of inefficiency are not obvious. In acute nephritis, for instance, renal function is more or less in abeyance, but we do not need a high degree of knowledge or technical skill to detect the fact; the condition is obvious. So, too, with subacute nephritis, passing on to the "large, pale" form of kidney, toxins are producing similar effects though less acutely; there is retention of water and of chlorides, with resulting raised blood pressure and a tendency to œdema and albumin can nearly always be demonstrated in the urine by ordinary methods.

What of the results of far more chronic infections, when the action of small quantities of toxins, long continued, produces gradual fibrosis or atrophy of renal substance, one or other effect predominating, but both present? The end of the process is death. Are we able to foresee that unhappy termination and perhaps postpone it even for a time? It may be that because of some symptom complained of we suspect that renal function is defective. Or perhaps some major surgical measure is contemplated and we wish to know whether the kidneys can stand up to an additional strain. We know from *post mortem* observations on previously healthy individuals that renal disease processes may make considerable headway without producing any warning symptoms; we know, too, that the reserves of a healthy kidney are enormous. Thus an increase of blood urea does not occur till renal efficiency is reduced to one-fourth or less.[1] To express it another way, it is estimated that a pair of healthy kidneys contain roughly ten million effective units and that one-fourth of this number working "full-time" can adequately perform the renal duties.

Herein lies the key to such success as we may attain with our present knowledge. If we can find evidence that the renal elements are overworking, that must be accepted as evidence that renal reserves are being called up because some of the renal mechanism has been destroyed.

Very often it is possible to do this. Probably the chief stimulus to increased activity of renal elements is an increase of glomerular capillary pressure which is usually reflected in the systemic circulation in an increase of arterial blood pressure.

I wish to stress this point. An increase of vascular tension, unless absolutely accounted for by some other factor, such, for instance, as thyreoid hyperactivity, should arouse a strong suspicion of renal degeneration, a process which we know to be steadily progressive. There may be no definite polyuria, albuminuria may be slight and even transient, nevertheless an increase of vascular tension even in quite early adult life should be most carefully watched and most seriously regarded.

We know that this is not infallible evidence of renal degeneration; we know also that in a later stage the pressure may fall to equivocal limits. By that time, however, other evidence is available. Deficiency of urea excretion, as shown by the usual test,[2] or of dye excretion which is a parallel test, may then be detected. A rise of blood urea indicates that the kidneys are indeed "far gone," while a high content of creatinine (so-called) in the blood, especially if independent of excessive meat intake, foretells an early fatal termination.[3] But our aim should be to discover the condition long before it has reached this stage. I believe that by carefully considering the vascular pressure together with the routine urinary examination and any clinical findings it is very often possible for us to do this and what is more important, even in the presence of a fairly satisfactory routine examination of the urine, a raised blood pressure should be accepted as presumptive evidence of renal damage until definitely proved not to be such.

The phrase "routine examination of urine" requires a little explanation. We usually record the specific gravity of the specimen being examined, but remembering that there is a fairly wide "normal" range, we may be at a loss how to interpret a reading, say, about 1014. As students we learned the importance of collecting the full twenty-four hours' secretion for quantitative work. This is also of importance in interpreting specific gravity readings. To go more fully into this portion of the examination, one may adopt the method of urinary test meal outlined by Mosenthal.[4][5] As Nye[6] has pointed out, Osler[7] states that this is undoubtedly the best test for early disturbance of renal function. Let me give here a few details from a reference[8] which should be exceedingly useful to the average practitioner:

If the standard diet cannot be employed, the patient may have three good meals in the day, one at 8 a.m., another at 12 noon and the last at 5 p.m. The bladder is emptied at 8 a.m. (after a night's fast) and the urine is collected at two-hourly intervals until 8 p.m. and the night urine is collected in one quantity from 8 p.m. to 8 a.m. The specimens are examined for volume, specific gravity, urea and salt concentration.

Normally the specific gravity will show variations as much as nine points or even more. In the nephritic individual one of the most noticeable points is the fixation of the specific gravity. The nephritic kidney tends to secrete a urine of persistently low specific gravity, despite the amount of food and water taken.

The normal night specimen is of moderately small volume (400 cubic centimetres) and high specific gravity, with a high nitrogen content, 1% or over. Renal impairment is indicated by loss of concentration in the night urine, associated with low specific gravity and nitrogen content.

There is also a tendency to total polyuria and the output of salts and nitrogen is decreased.

This method admittedly involves some trouble and may present difficulties in private practice; but even in its purely physical aspects it constitutes a court of appeal that will be of the greatest value in many early cases of renal degeneration.

Whether by noting the response of the kidneys to such a diuretic as caffeine it is possible to gauge roughly the effective renal reserve it is hard to say. The suggestion has been made[9] and as the action of caffeine is similar to the effect of raised vascular pressure, it seems reasonable to believe that we may thus obtain an indication as to whether the normal compensatory process can still go a good deal further or whether it has already almost reached its limits. The problem, however, is a complicated one.

In regard to treatment of these earlier stages of renal degeneration, I shall content myself by stating that as the condition is due to the long-continued action of toxins of various kinds, every effort should be made to discover whence these are originating. Nye[6] has called attention to the importance of lead as a probable cause of renal degeneration in many children in Queensland. Such a possibility must be kept in mind. Foci of infection must be diligently searched for and we must bear in mind that intestinal toxæmia is often an originating or an underlying factor. Much recent work on focal infection and on the determination of pathogenicity promises to help in the solution of the renal problem. Some day, perhaps, we shall be able to deal with these infections completely by general measures, but at present we must endeavour to trace the source of the trouble and clear it up.

Finally, as the significance of vascular hypertension is indeed a controversial matter, I would make one contribution to the discussion. The outcome of the foregoing observations is briefly this: that in general a finding of increased vascular pressure should be taken as an indication for a relentless search for a source of toxæmia somewhere in the body. What does it matter, then, whether the toxins have acted chiefly on the kidneys or predominantly on the vascular system itself? To quote our former master, the late Harry Brookes Allen:[10] "Excessive changes in one system will soon produce reflected changes in the other and a constant interaction takes place." Hence, whether the hypertension is due to changes primarily renal or primarily vascular, both systems will be affected to some extent and in any case the indications for investigation and treatment are the same.

I regret that the list of references is not so complete as it might be. The points of a discussion recorded in the journals are sometimes noted and the reference not retained. A number of excellent articles has provided some of the material embodied herein. I am indebted to Dr. Harold Moore for valuable assistance in respect to some of the references mentioned.

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PLASTIC RESTORATIONS AFTER SPECIFIC LESIONS.

By PERCIVAL PICKERILL, C.B.E., M.D., M.S.,
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THERE seems to be a prevalent idea that plastic restorations after losses of tissue due to syphilitic or lupus infections are not possible and that these unfortunates are doomed to go through life with repulsive features or to cover them up with dressings or some form of mask more or less crude and ill-fitting. Fortunately, however, this is not the fact and my experience has been that losses due to both these infections can be successfully treated surgically, provided that the lesions are in a quiescent condition and have received adequate previous treatment.

With regard to syphilis, it is not necessary to wait for the patient's serum to lose the power completely of reacting to the Wassermann or the Kahn test;

the reaction may be +−−. Indeed I have operated successfully on a patient whose serum yielded a ++− reaction, but never successfully on one whose serum yielded a +++ reaction; not that I have ever tried intentionally, but in endeavouring to account for the failure of some graft which in my opinion should have succeeded, a blood test has revealed a +++ Wassermann reaction. It may be inferred that such a situation should not arise and that one should always be sure on this point, but as a matter of fact patients sometimes intentionally mislead one and give such a definite history of some local infection or traumatism, backed up occasionally by a letter in all good faith from a doctor, that there seems to be no adequate reason for suspecting syphilis. Of course, the majority of syphilitic lesions of the face are unmistakable, but there are others which are so well healed, so clear cut and healthy looking that they would arouse no suspicions. However, without the most positive evidence of trauma I would not now embark on plastic restoration without a blood test, for there is no doubt whatever in my mind that grafts and positive Wassermann reactions are quite incompatible.

The following cases illustrate varying degrees and situations of nasal destruction and also various methods adopted in reconstruction.

Total Collapse of Nose.

The first patient was a girl aged thirteen years. Here the superficial tissues had scarcely been attacked, but the nasal bones and septum had disappeared entirely, with the consequent complete collapse of the nose, leaving it quite flat with the face (see Figure I). Owing to the cicatrized condition of the tissues from the intranasal aspect it was not possible to raise the integument, neither was it possible to remedy matters by the insertion of

FIGURE I.

cartilage grafts, since the skin of the nose had apparently ceased to grow with the rest of the face and was much too small in area. There were no signs of active infection and there was no reaction to the Wassermann test. A total rhinoplasty was therefore decided upon. This was carried out by the Indian method from the forehead, the consequent raw area being replaced by a whole thickness skin graft from the arm. The lower part of the skin of the old nose was utilized for lining the new alæ and rib

FIGURE II.

cartilage grafts were inserted subsequently for the bridge of the nose and columella. The result is seen in Figure II.

Total Destruction of Upper Third of Nose.

The patient was a woman, aged forty-three years. In this case skin, nasal bones, mucous membrane and septum

FIGURE III.

in the upper third had disappeared, leaving an ugly cavity. The tip of the nose had also been drawn upwards by the cicatricial contraction around the hole (see Figure III). The serum yielded a +−− reaction to the Wassermann test.

Partial frontal rhinoplasty was decided upon by the well known Valency swing method, thus obtaining a lining covered with cartilage for the hole. The frontal flap was swung down and skin was grafted on the raw area. About three weeks later an additional cartilage graft was inserted for the whole length of the nose in order to give a more decided bridge and also to depress the tip. The result is seen in Figure IV.

FIGURE IV.

The psychic effect in this case was so great as to be almost unbelievable. From being depressed and morose, she became vivacious and gay and her gratitude almost embarrassing.

Total Destruction of the Lower Half of the Nose and Complete Loss of the Upper Lip.

The third patient was a man, aged thirty-three years, who had lost the lower half of his nose and his upper lip

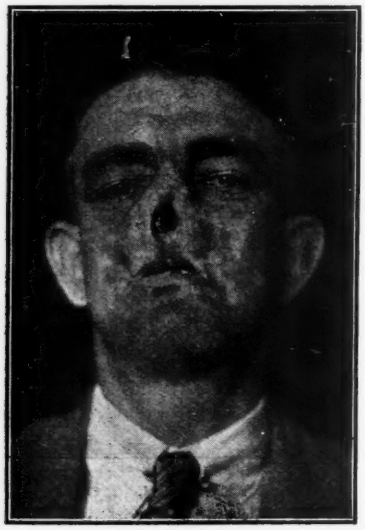

FIGURE V.

(see Figure V). There was no reaction to the Wassermann test. This case presented considerable difficulty owing to the dense scarring surrounding the lost areas and the consequently poor tissue upon which to graft; there was also the impossibility of using any of it for lining purposes. It being found impracticable to obtain sufficient skin from the forehead for a rhinoplasty, it was determined to obtain this from the abdomen. A totally new upper lip was also required and this was fashioned in the manner I have previously described as the double tube graft from the scalp and chest. The operations necessarily extended over a period of some months and can be summarized in stages thus: (i) A tube graft was fashioned from the abdomen, a double tube graft from the scalp and chest; (ii) a double tube graft was swung across to the prepared bed for a new lip; (iii) the distal end of the new lip was inserted into the bed, the proximal end of the scalp tube was returned, the abdominal tube was attached to the wrist, the cartilage was inserted into the upper end of the nose for the Valency swing; (iv) the tube was detached from the abdomen and inserted in the prepared bed in the upper part of the nose; (v) the tube graft was attached from the wrist and inserted in the prepared bed to form the lower part of the nose; (vi) several subsequent minor operations for shaping were required.

The result is seen in Figure VI. From being a recluse, wearing a mask, afraid to appear in public, the patient became an ordinary individual leading a normal life. He obtained employment without difficulty and he has since been married.

FIGURE VI.

Partial Destruction of the Tip of the Nose.

The course of treatment of the fourth patient had just concluded. He is a man, aged twenty-eight years. It will be seen from the accompanying illustration (Figure VII) that the tip of the nose and the anterior portions of both alæ have been lost by ulceration and that the surrounding tissue was cicatricial. His serum did not yield a Wassermann reaction. After due consideration it was decided that a lateral temporal flap would serve best for this patient. A tube graft was therefore fashioned containing the anterior superficial temporal vessels. It was swung down into position immediately and lined with a Thiersch pressure graft, the forehead raw area being covered with a whole thickness pressure graft. At the end of a fortnight the tube graft was divided, opened out and replaced, with the result seen in Figure VIII.[1]

[1] Other illustrations to this article could not be included owing to lack of space.—ACTING EDITOR.

If at all possible this method of restoration is excellent when the tip of the nose is involved. It will be noted from the illustration that the flap lends itself very well indeed to the shaping of alæ

FIGURE VII.

and columella. The excellent results obtainable, especially on the forehead, from the whole thickness pressure graft is also demonstrated.

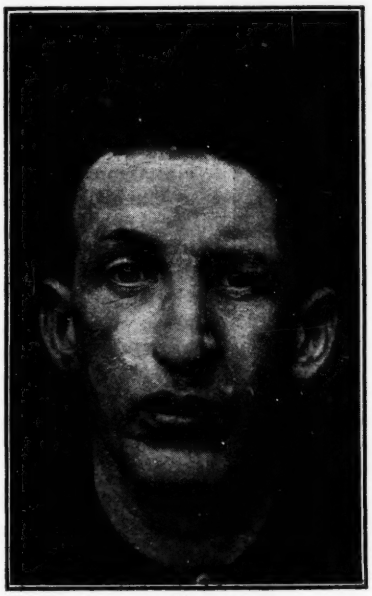

FIGURE VIII.

Reconstruction of the Tip of the Nose.

The reconstruction of the tip of a nose requires much more skin than one would imagine and this is not always available on the forehead, either by the Indian or the lateral-temporal method. If the case is not suitable for the abdominal method already described, it may be done by means of a tube graft taken from the neck. I showed wax models illustrating this method at the third session of the Australasian Medical Congress (British Medical Association) in Sydney last September. The method has the disadvantage that the lines of the returned tube graft in the neck are apt to become conspicuous by reason of keloid formation, but it has the great advantage of importing a good type of skin in the required amount, in a readily accessible manner and with little discomfort to the patient.

When only a small area is lost, such as a portion of one ala or the columella, a suitable graft may be obtained from the finger or wrist by the pedicle method.

In cases of superficial losses of the bridge or tip of the nose or in the early stages of lupus the area may be excised and restored by means of a free graft, whole thickness under pressure. Free grafts, however, more than pedicle grafts can be successful only if all active infection has ceased or been adequately controlled. With regard to extensive losses from lupus it is in my experience unwise to attempt plastic restoration until the process has been arrested by suitable light treatment. From a plastic surgery point of view lupus infected tissue which extends beyond the obvious ulceration, resembles exactly tissue which has been treated with radium, in that it is affected by a peculiar inertness and lack of response. Lupus in the early stages, however, may be most satisfactorily treated by excision and grafting either by the free or the pedicle method.

A NEW AUSTRALIAN GERMICIDE.

By E. Morris Humphery, M.B., Ch.M.,
Honorary Surgeon, Royal North Shore Hospital of Sydney.

An essential oil extracted from a variety of tea tree growing in profusion on the north coast of New South Wales was brought to my notice about twelve months ago. Analysis by Mr. W. R. Penfold, Curator and Economic Chemist of the Sydney Technological Museum, showed that it was non-toxic, non-irritating and eleven to thirteen times stronger than carbolic as a germicide (Rideal-Walker coefficient).

These properties suggested that it ought to be a valuable antiseptic for surgical use and I asked the manager of Australian Essential Oils, Limited, who brought the oil to me, to see if a saponified solution could be made so that it could be applied in various strengths of watery solutions for washing wounds, douching, dressings and other applications. His experiments were successful and the result is a pleasant smelling, clear solution of amber tint containing 35% of pure oil, readily miscible with water, forming a clean, white, milky looking fluid.

The results obtained in a variety of conditions when it was first tried, were most encouraging, a striking feature being that it dissolved pus and left the surfaces of infected wounds clean so that its germicidal action became more effective and without any apparent damage to the tissues. This was something new, as most efficient germicides destroy tissue as well as bacteria. Commencing with 2·5% dilution with water I found that quite strong solutions and even the pure saponified solution could be used without harm, the oil neutralizing the soap and preventing any caustic action.

Dirty wounds, such as are frequently seen as the result of street accidents, may be washed or syringed out with a 10% watery lotion, the solvent properties will loosen and bring away the dirt which is usually ground in, and the tissues will remain fresh and retain their natural colour. Dressings dipped in a 2·5% solution may then be applied, changed every twenty-four hours and healing will readily take place. Several times it has been possible to close dirty lacerated wounds immediately after cleansing in this way and they have healed by first intention.

The pus-solvent properties led me to try the lotion for perionychia which is usually so disheartening and so frequently results in the loss of the nail or deformity. Here the results were excellent. Infections which had resisted treatment of various kinds for months, were cured in less than a week. The fingers were dressed with lint soaked in 10% solution and changed every twenty-four hours, the patient being instructed to moisten the dressings with water if they became dry. On one occasion the patient in her anxiety to hasten the healing process applied undiluted lotion and the result was quite successful, the finger healing quickly and leaving the nail unharmed.

Photographs showing density of growth of *Melaleuca alternifolia* at Bungawalbin Creek, near Coraki, Richmond River, New South Wales.

Twenty drops in a tumbler of warm water used as a gargle quickly clear up a sort throat in the early stages; it should be an excellent prophylactic for many infective conditions which gain entrance to the body through the naso-pharynx.

Many other uses will probably suggest themselves, such as vaginal and intrauterine douches. In the vagina it can be used in much stronger solution than other similar preparations and advantage taken of its powerful germicidal action on account of absence of irritation.

As a constituent of ordinary hand soap its action on typhoid bacilli has been found to be more than sixty times as rapid as other so-called disinfectant soaps.

The smell of the oil is very pleasant and very like the smell of the tea tree growing in the bush. It is an excellent deodorant and immediately clears away any foul smell from a wound or abscess.

A few drops inhaled from a handkerchief will keep one free from attacks of coryza. I know of one sufferer who has enjoyed freedom from this trouble for a year, whereas previously he had frequent attacks for as long as he can remember.

Diluted with paraffin the oil makes an efficient spray for the naso-pharynx and as an ointment there are several parasitic skin diseases which should yield to it. As a constituent of wax dressings for burns it appears to promote healing, but I have not yet tried it sufficiently often to determine how to use it in this work.

Internally it should be of value in doses similar to those of the oils of the same class, eucalyptus, cinnamon *et cetera*.

The oil is obtained from the leaves of the variety of tea tree known as *Melaleuca alternifolia*. Cutting the branches away stimulates the growth and in less than twelve months new branches appear, giving a greater yield of oil than the original crop. The illustrations show the original growth and the new growth twelve months after cutting.

CALCIUM METABOLISM.[1]

By HENRY PRIESTLEY, M.D., Ch.M., B.Sc.,
Associate Acting Professor of Physiology, University of Sydney.

CALCIUM salts play a very important part in the animal economy; for growth and maintenance to

[1] Read at a meeting of the Orthopædic Section of the New South Wales Branch of the British Medical Association on July 4, 1929.

go on in normal fashion in animals not less than a certain minimum amount of calcium must be provided in the dietary. The adult human body contains about 2·5% of calcium and about 25% of the weight of dry bone is calcium.

Calcium is essential for the proper functioning of the nerves, muscles and probably all the tissues. Calcium salts are absorbed with difficulty from the intestinal tract and much more must be taken in the food than is required by the tissues.

Undoubtedly the best source of calcium is milk. The calcium content of human milk varies from about 0·02 to 0·04%. Cow's milk contains three to five times as much calcium as human milk, but the calcium is not so readily absorbed from the cow's milk as from human milk. The average assimilation of calcium expressed as a percentage of the intake as determined by a large number of investigators is 59 in the case of human milk and with cow's milk it is 25 in the case of infants and up to 40 in older children. Certain vegetables, such as carrot, spinach, cabbage, are also rich sources of calcium, although there is evidence that in children at any rate the calcium is less readily absorbed from vegetables than from milk. It is generally considered that an adult should have 0·7 to one gramme of calcium per day. Growing children need relatively more, for they are forming bones. In older children 1·0 to 1·5 grammes per day are necessary for proper development. In the case of infants some interesting observations may be made. For every hundred grammes of increase in the weight of a growing infant normally from 0·07 to 0·09 gramme of calcium should be retained in the body. If we take the calcium content of human milk as 0·03% and the retention from human milk as 70%, which is higher than usual, during the first two months of life, the demand for calcium is not covered by the intake. From the third month the balance will be established and then there will be an excess over the requirements. But the milk of many mothers contains less than 0·03% of calcium and the average assimilation is only 59%, so that the possibility of calcium starvation is very real. Rickety infants several months old not infrequently will have less calcium in the body than they had at birth. Here, as we shall see later, there is almost always deficiency of calcium in the diet and always deficiency in calcium assimilation.

Although cow's milk contains more calcium than human milk, the percentage assimilation is less, but whole milk would more than cover the requirements. The milk is, however, usually diluted, so that the amount available would be about the same as with human milk.

It is sometimes thought desirable to increase the calcium intake by giving free calcium salts. Many erroneous ideas are prevalent with regard to the use of these. The salts commonly employed are the chloride, lactate and phosphate, less commonly the carbonate. No really clear evidence has yet been brought forward to show that any one calcium salt is better than any other as far as availability is concerned. That calcium chloride is absorbed with greater ease than calcium lactate has been claimed because the serum calcium in normal individuals can be increased to a greater degree with the chloride than with the lactate. It is easy to show that this reasoning is quite false. When a sufficient dose of calcium chloride is given to have any appreciable effect on the serum calcium—and it needs a very large dose—a condition of acidosis is set up. Now one can produce a similar acidosis by the administration of ammonium chloride and here, too, there is an increase of serum calcium. The increased calcium in the blood after the administration of calcium chloride comes in the main from the tissues and not from the calcium administered.

For the proper absorption of calcium from the alimentary tract acid seems essential. This in general is, of course, supplied by the hydrochloric acid of the gastric juice. In the presence of the hydrochloric acid of the stomach contents calcium will practically all be present in ionized form and it will remain in this form as long as the reaction is on the acid side of neutrality. When the contents of the alimentary tract are predominantly alkaline, much of the calcium will be converted into insoluble calcium phosphate and calcium carbonate and hence be unabsorbed.

Carbohydrates and particularly lactose aid in the absorption of calcium probably because they are in part converted in the intestine into lactic acid. Phosphates in large amounts definitely decrease the absorption of calcium by converting the calcium into calcium phosphate. The most important factor in the absorption of calcium is the antirhachitic vitamin, vitamin *D*, about which I shall have more to say later.

Like all other inorganic salts calcium salts are continually being excreted from the body. Some is contained in the urine, but the greater part is probably excreted into the large intestine. Just what are the relative proportions it is impossible to determine.

Bergeim has shown that calcium is absorbed in the small intestine and excreted into the large intestine. In rickets calcium is absorbed from the small intestine, but is excreted into the large intestine in excessive amounts. Apparently the blood cannot retain the absorbed calcium.

Calcium salts circulate in the blood almost wholly in the plasma and the amount is remarkably constant in health and in most diseases. The average calcium content of the serum is ten milligrammes per hundred cubic centimetres, ranging from about 9·5 to 10·5. It exists in the blood in three forms, part ionized, part not ionized but dialysable, part unionized and undialysable. The relative proportions of these three forms varies under different conditions and there is no general agreement yet as to what are the usual figures. The greatest physiological variation in the serum calcium occurs in pregnancy. Corresponding to the rapidly increasing

demand for calcium by the growing fœtus the content of calcium of the mother's serum tends to fall.

It is very difficult to raise the calcium content of the blood, when the figure is normal, by the oral administration of calcium salts, except, as we have seen, with large doses of calcium chloride; the effect quickly passes off. When the blood calcium is for some reason lower than normal, it can be raised by oral administration, but to have any permanent effect the cause of the lowering must be sought for and treated.

The administration of parathyreoid extract will cause a considerable increase in serum calcium apparently by mobilizing tissue calcium and College has described a condition of hypercalcæmia which might be fatal.

Ossification of Bone.

One of the most interesting aspects of calcium metabolism concerns the ossification of bone. Bone consists largely of a protein matrix, osteoid, which is impregnated, generally in very regular fashion, with phosphates and carbonates of calcium and to a very much less extent of magnesium. Calcium accounts for 37% of the ash of bone and this percentage persists whether the bone be healthy or affected by rickets, osteoporosis or osteomalacia. This, of course, suggests a fixed composition for the mineral constituent of bone. What this substance is has not yet been settled; it is not tricalcium phosphate, but appears to be a complex salt of the type of the mineral apatite. It is sufficient to say that it contains calcium, phosphoric acid and carbonic acid. Many views have been held as to the mode of deposition of calcium salts in ossifying bone. The most recent and most satisfactory view is that put forward by Robison. An examination of growing bone shows that it contains an enzyme capable of splitting up hexosephosphates. When extracts are made of growing bone, these extracts, acting on soluble calcium hexosemonophosphate, cause the separation of insoluble calcium phosphate. The enzyme can be shown to be present in the bone itself and to bring about this separation *in situ*. In rhachitic bones the osteoid tissue, in which the calcium salts are normally deposited, is formed, but the calcium salts are not deposited. Extracts of these bones are, however, active in hydrolysing hexosephosphates and if a rhachitic bone be placed in a solution of calcium hexosephosphate, insoluble calcium salts are precipitated in just those places where they would occur if the bone were normally calcified. The thing lacking for the proper calcification is not the bone enzyme, but probably some abnormal relation between calcium and phosphorus in the blood bathing the tissues. Blood contains hexosephosphates which are attacked by the bone enzyme, although they exist mainly in the red corpuscles which possibly act as a reservoir, keeping the content of the plasma relatively constant in health. The bone enzyme is not found in unossified cartilage and in partially ossified bone it is found only at the ossification centres. The full details of the process are not yet clear, but there seems considerable justification in Robison's claim that "the production of the enzyme is a part of those cellular activities which result in the formation of bone."

This leads us to the very important studies on calcium metabolism which have been made during the past few years in rickets and allied conditions.

It will not be out of place here to direct your attention to the normal process of ossification in bone and the departures from the normal in rickets and similar diseases. In the case of a long bone the growth in length takes place between the shaft of the bone and its head or epiphysis. In the fœtus the bones are laid down in cartilage, later an ossification centre appears and the shaft of the bone is calcified, the cartilage being replaced by calcified osteoid. Cartilage still continues to be formed at the ends of the bone. Later separate ossification centres commence at each end, forming the epiphyses, but between these areas of calcification in the epiphyses and the calcified shaft cartilage continues to be formed, to be replaced by osteoid and calcified. In the zone of cartilage immediately adjoining the calcified bone the cartilage cells become arranged in columns. This is "the zone of proliferative cartilage." It would take too long to go further into the histological changes leading up to the final ossification. Growth in thickness of the bone takes place by the laying down of osteoid and its calcification under the periosteum with a progressive absorption of the first formed bone to form the larger marrow cavity of the larger bone.

On this question of absorption it is to be remembered that calcium metabolism in bone is dynamic; absorption is always going on in some part and calcification in others throughout life.

Calcium and Rickets.

In the rhachitic bone all the processes are abnormal. In the normal bone the junction of the bone with the cartilage forms a regular line. In the rhachitic bone the line is ragged. The columnar arrangement of cartilage cells is absent and the cartilage is replaced by uncalcified osteoid in irregular manner so that islets of cartilage persist. There is no provisional zone of calcification. Abnormal amounts of osteoid are produced in the endeavour to compensate for the weakness of the bones.

Rickets occurs only in growing bone. In the full grown bone there is no cartilage except articular. The analogous conditions in the adult are osteoporosis and osteomalacia.

In the ætiology of rickets there are three principal factors concerned, calcium, phosphorus and vitamin *D*. With regard to calcium and phosphorus it is rather the relative proportion of these two in the dietary than the absolute amounts that is important. Rickets can be produced when the diet contains normal or excessive amounts of calcium but deficiency of phosphorus, and also with normal

or excessive amounts of phosphorus and deficiency of calcium, provided that there is also at the same time a deficiency of vitamin *D* or its equivalent, ultra-violet light. If there is a sufficiency of vitamin *D* in the diet, neither calcium nor phosphorus deficiency will lead to rickets, but osteoporosis may appear. Human rickets occurs only in the growing child and most commonly in the first year of life when milk forms the sole or main diet. I have pointed out previously that there is grave danger of calcium starvation during this period. The more rapid the growth, the more likely is rickets to appear with food deficiencies. It is probably because of this that premature children so often get rickets. If growth ceases for any reason, as starvation, calcification proceeds in the abnormal bone.

The rickets produced in animals by low phosphorus and normal or high calcium in the diet is characterized by an histological condition corresponding to that ordinarily found in the human being. Further, there is a low phosphorus content in the blood and a normal calcium content, the condition usually found in uncomplicated rickets in children. When the calcium is low and the phosphorus high in the diet the histological picture is somewhat different; among other things the arrangement of the cells in the metaphysis is more orderly. In these conditions the calcium of the blood is low and the phosphorus normal or little reduced, a condition found in children in whom rickets is complicated with tetany. Exactly similar relations are found in osteomalacia.

When there is a low content of calcium in the blood tetany is very likely to appear, the tetany being an expression of the nervous tissues of an insufficiency of calcium ions, just as rickets is an expression on the part of the skeleton of disturbed relations between calcium and phosphate ions in the body fluids.

The addition of magnesium carbonate to the diet and also strontium carbonate can bring about very severe rickets. The magnesium carbonate is most effective in diets in which the phosphorus is low. Magnesium carbonate is then not a drug to be given to children with rickets or any suggestion of deficient calcification of bones.

If there is a sufficient amount of calcium and phosphorus in the diet, rickets does not result even with great deficiency of vitamin *D*, but osteoporosis may be seen.

The calcium content of the bones reckoned on the dry weight has been used as a criterion of the presence of rickets, but owing to the high proportion of fat in bones showing osteoporosis, the calcium appears low in these and a better comparison is the calcium content of fat-free bones. Chick, Karenchevsky and Roscoe suggest what they call the A : R ratio as a criterion. If the sum of the weights of the water, fat and ash be subtracted from the weight of the fresh bone, what they call the "organic residue" remains. This amounts to from 20% to 22% in bones of rats, whether normal, osteoporitic or rhachitic. The A : R ratio is the ratio of the bone ash to this organic residue and it is much used now by English investigators in describing their results. In normal rats the A : R ratio is in the neighbourhood of 1·5; in osteoporosis usually from 0·9 to 1·2; in rickets from 0·4 to 0·8.

Vitamin D.

A point of very great interest is the probable relation of cereals in the diet to rickets and deficient calcification generally in bones and teeth. E. and M. Mellanby have done a considerable amount of work on this during the past few years and have adduced some very striking evidence. There appears to be in cereal grains some substance which acts in the opposite sense to antirhachitic vitamin. Different cereals differ quantitatively in their effects. Oats and oat products, oatmeal, groats and so forth have by far the most intense action. In Mellanby's experiments white flour had the least action of the cereal products tested. Rice and maize occupied intermediate positions. Foodstuffs rich in antirhachitic vitamin oppose to different degrees the rickets-producing effects of cereals. Cod liver oil is so potently antirhachitic that it can easily antagonize the rickets-producing effects of cereals, even oatmeal. Yolk of egg is also powerful. Butter is poor but working with the remaining constituents of milk or when calcium salts are added to the diet the effect of butter is greatly increased. Calcium salts alone antagonize the rickets-producing effects of cereals to a certain extent, but oatmeal contains much more calcium than the other cereals. Ultra-violet light is potent in overcoming the bad effects of cereals for reasons that are now obvious. The nature of the deleterious substance or substances has not yet been determined.

M. Mellanby has shown that cereals also interfere seriously with the calcification of the teeth. Now some interesting questions arise out of this. Cereals are becoming more and more the principal articles of diet of man. Further, fine races of men have been raised on diets of which the worst of the cereals, oatmeal, forms a large part. The diets of these people also included foods rich in antirhachitic vitamins, eggs, milk, fishes of the fatty variety.

In tropical countries where cereals form such a large part of the diet, the sunlight no doubt plays a large part. This is well seen in the very great prevalence of rickets among young girls in some parts of India and osteomalacia among certain classes of women in China. In India, with child marriage, girls go into *purdah* at a very early age. Thereafter they live in semidarkened rooms and never see the sun. Cereals, of course, form a large part of the dietary. In China a very advanced form of osteomalacia is not uncommon in certain districts and among women of a certain class, the wives of better class farmers. They do no field work and spend most of the day resting on warmed couches in semidarkened rooms, taking no exercise. Cereals, in the form of millet, form a very large part of the

diet and antirhachitic vitamin-containing foods are scarce. Osteomalacia developes, the appetite, never good, becomes poorer and a vicious circle is set up. The blood phosphorus is very low in these people and in some the blood calcium also.

A large amount of cereal in the dietary of many people in cities with the rest of the diet poor in antirhachitic vitamin and calcium, probably accounts for the very great prevalence of rickets in such murky places as Glasgow.

In Australia the abundant sunshine should overcome much of the deleterious effects of poor dietaries. This leads us to the question of antirhachitic vitamin.

It is not necessary to say anything here concerning the distribution of antirhachitic vitamin in natural foodstuffs. More interesting is the nature of the substance and the relation of radiant energy to it. That ultra-violet light had a curative effect in rickets has long been known, but it is only recently that the nature of this effect has been known.

On certain diets rats developed rickets when kept in the dark or in a room to which direct sunlight had not access. When rats on the same diet were exposed to direct sunlight for only a short time each day, they did not develope rickets. The same thing was shown in leg weakness in chickens. When the animals were exposed for a few minutes each day not to sunlight, but to the radiations from a quartz mercury vapour lamp or other artificial source of ultra-violet light, they did not develope rickets. When the food was radiated and not the rats, again rickets did not develope. A large number of foodstuffs were then irradiated and many of them became antirhachitic although previously they had no such action. Investigation showed that a substance common to all the foodstuffs was cholesterol. Further, when cod liver oil was saponified the antirhachitic property was found to remain in the very small part which was unsaponifiable. This part consists largely of cholesterol and other sterols. Cholesterol when prepared from some such source as brain had no trace of antirhachitic activity, but if the cholesterol were exposed to ultra-violet radiations, it became actively antirhachitic. It was at first thought that some change took place in the cholesterol as the result of the radiation, but this was doubted by some observers and some impurity was looked for. Finally Rosenheim and Webster in England and Windam in Germany working in collaboration showed that the impurity which goes with the cholesterol obtained from all the sources examined and from which vitamin *D* is formed, is another sterol, ergosterol. Pure ergosterol has been prepared in quantity from yeast and when this substance is irradiated, it becomes intensely antirhachitic. Rosenheim and Webster showed that the limiting prophylactic and curative dose of irradiated ergosterol for rickets in rats is of the order of 0·0001 to 0·00005 milligramme per day. Dr. Katherine Coward has even been able to demonstrate in rats a definite calcifying effect of 0·00001 milligramme of irradiated ergosterol. As approximately 10% of the ergosterol is probably changed, the amount of artificially produced vitamin *D* which has an effect, is astonishingly small. Human rickets is rapidly cured by doses of two to four milligrammes per day of irradiated ergosterol.

Five milligrammes of irradiated ergosterol are equivalent to about one litre of good cod liver oil. The exact nature of the substance produced by the irradiation has not yet been determined, but it is evidently a sterol.

Human skin contains 13% to 24% of sterols including ergosterol, so that there is no difficulty in accepting the view that the action of ultra-violet light, applied directly to animals, is due to the formation of vitamin *D* in the skin and its subsequent transference to other parts of the body.

It is interesting to note that recent work has shown that a condition of hypervitaminosis can be set up in animals by giving excessive doses of irradiated ergosterol. There are then apparently two thresholds for vitamin *D* minimum and maximum. When more than a certain amount is given, some 100,000 times the minimum effective dose, animals die with relatively enormous deposits of calcium in the heart muscle, kidneys, arteries and other organs and urinary calculi are often found in the bladder. The blood phosphorus content also becomes remarkably high and frequently also the blood calcium content.

Reports of Cases.

CONGENITAL RUPTURE OF JEJUNUM.

By A. E. Harrison Salter, M.B., Ch.M. (Sydney),
Blayney, New South Wales.

Clinical History.

A woman, thirty-eight years old, was pregnant for the sixth time. The membranes ruptured at 9 a.m. and a vaginal examination showed that the presentation was vertex and the position right occipito-posterior. The cervix was about half dilated. Rotation was performed manually and dilatation was completed by the same means. Forceps were then applied and the child delivered at 1 p.m., the whole procedure giving no trouble whatever. The child appeared to be normal, breathed well and cried lustily. The placenta was delivered twenty minutes later and was quite normal. The subsequent puerperium was entirely uneventful.

The following day the child appeared normal, except that no meconium was passed and during the evening two cubic centimetres of castor oil were given. The next morning the child showed signs of cyanosis and as no meconium had yet been passed, a bowel wash-out of normal saline solution was given and the fluid returned quite clear and uncoloured. There was a history that the first child had had "obstruction to the bowel" and the parents became very worried about the present infant, suspecting a similar condition.

A barium enema was then given and was observed to flow freely to all parts of the large bowel as far as the ileo-caecal junction and no abnormality was detected

other than that the lumen of the colon appeared small, being only about 0·5 centimetre.

A barium meal was then given and observed to fill the stomach normally. The pylorus remained closed and the examination was deferred for a time to allow the barium to pass the sphincter.

The child's condition, however, became worse. The cyanosis increased and the child died quite suddenly when exactly forty-eight hours old and before further X ray examination could be made.

Post Mortem Findings.

On opening the peritoneal cavity from xiphisternum to umbilicus a large quantity of fæcal fluid escaped having a most offensive smell and the appearance of a watery enema result as obtained from an adult. Examination of the large bowel confirmed the X ray findings, it being small in lumen and thick-walled.

The small intestine was somewhat dilated in lumen throughout, a Meckel's diverticulum was observed and in the jejunum about fifteen centimetres from the pylorus there was a portion of bowel about two centimetres in length which was blackened, gangrenous and friable, there being a large area of it broken through and much fæcal fluid escaping therefrom.

The mesentery in that region was quite normal. The arterial and venous network were carefully examined for evidence of thrombus or embolus, but nothing was seen to suggest this, the blood in the vessels being observed to be unobstructed in its flow in either direction.

The portion of the bowel affected was easily found and brought out of the abdomen and no constricting bands or other causes of strangulation could be discovered.

The stomach, pyloric sphincter and all other viscera were examined and found to be normal.

Comment.

The mother volunteered the information that she had fallen flat upon her face five days before the child's birth and indeed her knees were both considerably discoloured by bruising as a result of the fall. The parents were quite satisfied with the explanation given, namely, that the aforesaid fall had injured the child's abdomen, it having been in the right occipito-posterior position, leaving the abdomen exposed.

Their first child had died in the same way, having lived almost exactly the same length of time, forty-eight hours, and it had vomited meconium-like substance and passed nothing *per rectum*. The parents said that they were told the child had had "an obstruction to the bowel" and the mother said she had had a fall before that confinement too!

Now although the aforesaid explanation fully satisfied the parents, one would be interested to get a bit nearer to the real cause and though I am unable to find records of similar cases, there must have been many such and I would be glad to hear anything that is known about them. I cannot see how either obstruction to blood supply or strangulation or any extraordinary form of small bowel intussusception could have been the cause.

A CASE OF IANTHINOPIA.

By MAX YUILLE, M.B., Ch.B. (Melbourne), F.C.S.A.

THE following case, brought under my notice by Dr. Keith Colquhoun, may prove of interest to all practitioners as well as ophthalmologists.

A.B., a male, aged eighty-three years, suffered from failing heart with œdema of the extremities and generalized *eczema senilis*. He was treated with *pilula diuretica* (containing *pulvis digitalis* 0·06 gramme or one grain) three times a day, commencing on October 16, 1929. On November 6, 1929, he first complained of violet-coloured vision; the whole field of vision appeared violet, though there was no apparent diminution of visual acuity.

The pill was discontinued and by January 3, 1930, the violet tinge, according to the patient, was much less pronounced. By January 17 the condition had disappeared.

Fuchs[1] mentions such cases as due to digitalis or disease of the choriold or retina. As the condition cleared up with the discontinuance of the pill, it would seem that disease of the choriold or retina might be excluded. No routine examination of the *fundus oculi* or test of visual acuity was possible.

Reference.

[1] H. E. Fuchs: "Text Book of Ophthalmology," Eighth Edition, page 247.

Reviews.

MEDICINE.

THE text book of medicine edited by Dr. Russell L. Cecil may be recommended as one of the best of its kind.[1] The editor has as his associate for diseases of the nervous system Dr. Foster Kennedy, Professor of Neurology at Cornell University, and he has availed himself of the assistance of a large number of able coadjutors. The names of many of these are well known in Australia and each of them has claims to be regarded as an authority on the subject matter of his contribution. Thus H. A. Christian and O'Hare write the article on nephritis and Cary Eggleston those on diseases of the myocardium and the treatment of cardiac failure. Ramsay Hunt, Noguchi and Allen B. Krause contribute the articles on *paralysis agitans*, yellow fever and tuberculosis respectively. Professor Peterson, of Columbia University, writes in a very interesting manner on the neuroses and in discussing neurasthenia he stresses the value of sodium glycerophosphate, states that he regards the Weir Mitchell rest cure as unnecessary and condemns the use of psychoanalysis. Altogether the selection of the various writers has been made with much discrimination and there seems also to have been a very careful editing, the volume as a whole being distinguished by its readability and clarity and for the absence of mannerism and fads.

Many of the articles include a brief but interesting history of the disease under discussion. References to leading authorities on the subject matter are fairly numerous. When prescriptions are given, they are written in the metric as well as in the imperial measure. Laboratory methods are frequently mentioned and methods of investigation are at times given in some detail, as for example in the elucidation of symptoms of diseases of the nervous system, in the examination of the *fundus oculi* in general diseases, in the method of lumbar puncture and the information to be gained from examining the cerebro-spinal fluid. Exact instructions are also given for such procedures as vaccination, intubation, tapping the pleura and the administration of serum in dysentery and pneumonia. As might be expected there are occasionally evidences of differences between the American and the Australian points of view. There is, for example, no mention of hydatid disease of the lung as a cause of hæmoptysis and we see no reference to the Casoni method of diagnosis of that disease. The probability of rabbits which are eaten by dogs, acting as agents for the dissemination of hydatid disease is not believed to be important here. It seems strange to see no mention made of the value of leeches in relieving pleuritic pain and to read that a combination of sodium salicylate and sodium bicarbonate sometimes seems beneficial in acute rheumatism. Many Australian surgeons, following probably the British viewpoint, would disagree with the statements

[1] "A Text-Book of Medicine," by American Authors; Edited by Russell L. Cecil, A.B., M.D.; Associate Editor for Diseases of the Nervous System, Foster Kennedy, M.D., F.R.S.E.; 1927. Philadelphia: W. B. Saunders Company; Melbourne: James Little. Royal 8vo., pp. 1522, with illustrations. Price: 45s. net.

that tuberculosis of the cervical glands is better treated by heliotherapy and X rays than by operation, as they would also with that of the estimate of the prevalence of syphilis among the general population as being less than 10%. Warthin, of Ann Arbour, is in agreement with the late Harry Brookes Allen that the proportion stated is too low. Then again we are accustomed to think that hæmorrhage in enteric fever bears some relation to the severity of the disease, that the pain in *angina pectoris* is presternal rather than præcordial and that functional albuminuria is not more common in girls than in boys. We are also inclined to place more importance on the alkaline treatment in *Bacillus coli* urinary infections than is done in the article in this volume. It is obvious that these are all matters on which there may well be differences of opinion and that a certain amount of dogmatism is expected in a students' text book.

It is interesting to note the condemnation of santonin as a remedy and also the recommendation of pumpkin seed rather than male fern as an anthelmintic for tape worm. The term remittance (instead of remission in fever—Malta fever) is unusual with us, as is the application of the term *bruit de diable* to the murmur heard over the thyreoid gland in exophthalmic goitre. There are a few typographical errors: Von Graeffe, page 1351, Foralini, page 853, Werikebuch, page 1068; the name of one author is given variously as Wichman, Wiekmann and Wickman. On page 1099 milligrams is evidently a misprint for grammes.

On the whole it must be said that the volume is an extremely valuable one and deserves to be widely read both by students and practitioners. It is fairly certain that subsequent editions will be called for and we hope that the high standard set up will be maintained or even improved upon in the new publication.

THE THYREOID GLAND.

"DISEASES OF THE THYROID GLAND," by Dr. Hertzler, is a book[1] of medium size and not as full of detail as that of Crotti, though the book is readable enough on account of the author's robust views and style. In the discussion of normal and morbid anatomy he takes little notice of the work of Williamson and Pearse. He is sceptical of rapid morphological changes. For example, he found no hyperplasia in the glands of executed criminals, though these should have strong emotional states. But do they? Goitres are classified anatomically into four main types: (i) colloid, (ii) adenomata without toxic symptoms, (iii) adenomata with toxic symptoms and (iv) the goitre of Graves's disease (exophthalmic goitre). The dictum on page 41 that these classes do not represent separate diseases, but merely stages at least for the most part of one progressive disease, is opposed to the views held by some authorities in Australia. Nor is the statement on page 43 acceptable that with the development of accessory acini in colloid goitre the gland becomes irregular and bosselated and is then called an adenoma. He admits that no one seems to know when the changes cease to be hyperplastic and become adenomatous. This point of view is not uncommon in the literature of goitre and seems to be the result of a large proportion of endemic goitre material in the location. He makes no sharp distinction between toxic adenoma and Graves's disease; it is but a question of severity of symptoms. It is called primary Graves's disease when the preexisting (*sic*) goitre was not discovered before toxic symptoms began.

The description of the morbid anatomy is good and the photographs are excellent. Figure LXV on page 89 appears to be a large adenoma and not, as the legend runs, a colloid goitre. Carcinomata take origin from fœtal adenomata and fall into three classes: (i) clinically benign, microscopically malignant, (ii) clinically and microscopically malignant, (iii) clinically malignant, microscopically benign. Fœtal adenomata should be resected for fear of malignant changes. He has never felt satisfied that he has cured a malignant goitre. Glands that are hyperactive, are not malignant, therefore toxic symptoms preclude the existence of malignant disease. The histology is well illustrated. Papillary tumours are placed among the carcinomata. The symptomatology is dealt with in detail. Little is said about auricular fibrillation. The chapter on diagnosis is well illustrated. In regard to treatment the outlook is surgical. Goitre is a progressive disease which the physician can only ameliorate. X ray treatment is condemned. Bosselated (nodular) goitres sooner or later kill the patient, if not treated surgically. Basedow goitres may burn themselves out after years of invalidism, occasionally end in myxœdema, but most of the patients die if not treated surgically. Extensive surgical resections almost regularly end in recovery. In only one patient out of one hundred is a second operation necessary! Surgical myxœdema should not be dreaded, as it is better to take thyreoid gland by the mouth than to be "encumbered with a defective tissue." The robust optimism of this surgeon is illustrated by a quotation (page 179): "Timely operation in goiter is as important as timely operation in cancer. Doping goiters of certain well recognized types with medicine rivals in wisdom the application of salve in cancer of the breast. It is confidently to be expected that some day the causative problems underlying goiters will be so well understood that surgical operations will not be necessary, but that time has not yet arrived." The book ends with chapters on goitres in unusual situations, hospital management of patients in surgical wards and the topographic anatomy of the thyreoid gland.

[1] "Diseases of the Thyroid Gland," by Arthur E. Hertzler, M.D., with a chapter on Hospital Management of Goiter Patients, by Victor E. Chesky, M.D.; 1929. St. Louis: The C. V. Mosby Company. Royal 8vo., pp. 286, with illustrations. Price: $7.50 net.

ANATOMY IN PICTURE FORM.

"BAILLIÈRE'S SYNTHETIC ANATOMY," by J. E. Cheesman, is the outcome of much ingenuity and hard work.[1] It is an atlas arranged in twelve parts, each part demonstrating very lucidly about twelve different stages of dissection. These dissections are reproduced on transparent paper. For instance, in Part IV there are reproduction of twelve dissections of the thigh and hip. The first picture of this series is that of the sensory areas of the gluteal region and back of the thigh on the right side; on the left side the skeleton of this area is demonstrated. In the second drawing the superficial fascia has been removed, revealing the cutaneous nerves and the segments of the cord from which they came. The third drawing shows the *gluteus maximus* muscle and the posterior cutaneous nerve of the thigh. In the fourth picture the *gluteus maximus* muscle and the deep fascia have been removed so that the *gluteus medius* muscle, *pyriformis* muscle, *obturator internus*, gemelli muscles the hamstring muscles, the sciatic nerve, the gluteal vessels *et cetera* are shown. This scheme is continued till in the deepest dissections possible from the back the femur is exposed posteriorly in its whole course and the deeper structures are seen; then by reversing the atlas and following the different drawings the student is led from the deepest dissections on the anterior aspect by gradual stages to the last one in the series which illustrates the sensory areas on the anterior aspect of the thigh and hip.

By superimposition of one drawing on another the relations of superficial structures to deeper ones can be readily appreciated. It is an excellent work both for teacher and student of anatomy, also for the busy operating surgeon who wishes to refresh his memory in regard to the relations of structures without having to turn up page after page of references. The drawings lend themselves to reproduction by tracing, also to enlarging, a fact that is very useful to the student of anatomy.

[1] "Baillière's Synthetic Anatomy," by J. E. Cheesman; Part IV: The Thigh and Hip; 1929. London: Baillière, Tindall and Cox. Crown 4to., 12 plates. Price: 3s. net.

The Medical Journal of Australia

SATURDAY, MARCH 29, 1930.

A New Milestone.

THE Commonwealth of Australia on account of its extent and of its situation in the southern part of the Pacific has peculiar responsibilities in connexion with public health. Its climate varies from temperate to tropical and its population, with the exception of persons congregated in a few large cities, is scattered over enormous areas. It is the trading centre for many of the islands of the Pacific and of those just beyond its northern shores. With their wealth of population, their endemic diseases and the susceptibility of their inhabitants to disease of the white man, these islands convey both a danger and a responsibility. They also offer an opportunity for research which in many respects is unrivalled. It was a realization of these facts which led that far-seeing and wise statesman Thomas Anderson Stuart to take the initial steps more than a quarter of a century ago in the founding of a school of tropical medicine. He enlisted the sympathy of other men of vision and set the movement on its feet. In view of its position as commercial and shipping centre he chose Sydney as the site of his school. But this was not to be. It is a matter of history, traced on previous occasions in these pages, how Anderson Stuart's original scheme was changed and the School of Tropical Medicine was established at Townsville. Subsequent events have shown, however, that he was right and his judgement was vindicated on March 6, 1930, when the School of Public Health and Tropical Medicine was formally opened at Sydney by the Honourable Frank Anstey, Minister for Health of the Commonwealth.

It is necessary to draw attention once again to the responsibility of the Royal Commission on Health for the inauguration of the new school. The Commission issued its report in January, 1926. Under the caption "Chairs of Preventive Medicine and Tropical Hygiene" the following statement was made:

A scheme has been submitted to us for the endowment by the Commonwealth of chairs and schools of preventive medicine and tropical hygiene in the Sydney University. We are of opinion that these schools should be established, but consider that at the present juncture it would be sufficient to provide a chair of preventive medicine with an adequate department of tropical hygiene which, if necessary, could be later expanded into a chair. The arguments put forward in favour of the selection of Sydney University for this purpose are to us convincing.

One feature in the work of schools of tropical medicine or hygiene should be the collection and condensation of the records of the medical administration of the Pacific Islands with a view to their distribution. Valuable reports from the Pacific Islands are wasted, as there is no central place at which they can be correlated and collated.

Among the recommendations was one to the effect that:

Legislation should be passed by the Commonwealth Parliament to provide university and other training for experts in public health, also for the training of the technical personnel for all public health services both in the Commonwealth and in the States.

The ceremony of March 6 was the direct outcome of the recommendations of the Commission. At the same time the recommendations have not been carried out in their entirety. The school of tropical hygiene is an accomplished fact, but the chair of preventive medicine has been neglected. The Minister for Health struck the right note when he stated that the school had been founded not merely for the issuing of diplomas, but for the teaching of preventive medicine and for research into the causation of disease. The work which was begun in Townsville, will be continued in Sydney and will be expanded. As Professor J. C. Windeyer, the Dean of the Faculty of Medicine of the University of Sydney, pointed out, the activities of the school will include the teaching of undergraduates, the instruction of graduates in public health and tropical medicine and research. The school as at present constituted will have the confidence of the medical profession and of the public. Those in authority have shown wisdom in the choice of a

director. Dr. Harvey Sutton can be looked to with assurance to lead his team of workers along the right path and to set a high standard of endeavour. The watchword will be prevention. It is interesting to note that already the Senate of the University of Sydney has given its approval to the suggestion that the school should exercise a general supervision over the health of the undergraduates, although as far as the undergraduates are concerned, the control will be optional. The financial responsibility for the school will rest with the Commonwealth Government and it will be under the control of a council composed of representatives of the Government and the Senate of the University. There will be no parochialism. This school is not a school of the University of Sydney, it is a school for the whole Commonwealth. It is destined to spread its influence over the Commonwealth, to the islands of the Pacific and beyond. At the same time its establishment does not mark the end of the journey, it is merely a milestone on the road of progress. The next milestone will be reached with the creation of the long-overdue chair of preventive medicine.

Current Comment.

PRECANCEROUS CONDITION OF THE BREAST.

It is not always realized that the breast is subject to constant stimulation. From the time of puberty until the menopause the breast is influenced by the hormonal changes occurring with each menstrual period. This may be recognized by the tenderness produced in the breast at each menstruation. This, of course, is more pronounced in some women than in others, in the same way as the general effects of menstruation present wide variations. The constant stimulation of the breast is regarded by Keynes as being of basal importance in the production of the condition known clinically as chronic mastitis. He pointed out in a communication on the subject which was discussed in these pages, that chronic changes in the breast are commoner in women who have not borne children than in those whose breasts have secreted milk. The products of cellular activity in non-lactating breasts must be absorbed from the ducts. In the lactating breast there is an opportunity for thorough drainage of the ducts to take place. Periodical lactation is the normal occurrence in woman. Since the lactiferous ducts are the site of periodical stimulation, it is but natural to expect that variations in the normal growth and structure will be manifested in this situation and this is what actually occurs.

Alexander A. Charteris has recently made a study of the changes in the mammary gland which precede the occurrence of carcinoma.[1] He comes to certain important conclusions, some of which carry an added interest in view of earlier work on the subject. He has examined fifty breasts, but has excluded two of them, since in one the tumour was a sarcoma and in the other it was benign. He found that the earliest evidences of epithelial activity are closely connected with so-called chronic cystic mastitis and he proceeds to discuss this condition. In all but seven of the breasts examined it was present in some portion of the organ. Occasionally it was found only on microscopical examination. Charteris states that the substance of the breast which is tough and fibrous, is often beset with numbers of cysts which may vary greatly in size. Sometimes the cystic change is to be found only on microscopical examination. Channels are seen filled with necrotic cellular material and retained secretions. Papillomatous growths are often seen in cysts or ducts. On microscopical examination the acini are seen to be the elements most affected. The change is due partly to the distorting influence of newly formed connective tissue and partly to abnormal epithelial proliferation. Some of the acini undergo proliferation and budding. It is not this change, however, which Charteris regards as of special importance, but changes in the epithelial lining of acini which already exist. The first suggestions of epithelial activity are usually found in connexion with so-called cystic mastitis. As a rule this occurs in the smaller ducts, but occasionally in acini which are dilated. Charteris found papillomata in a considerable proportion of breasts affected in this way; sometimes these channels constituted a way by which intraduct carcinoma grew towards the surface. Epithelial hyperplasia in the finer divisions of the ducts usually resulted in the formation of more or less solid sheets or masses of cells, but here papillary branching and adenomatous arrangements were observed on a smaller scale, so that there did not appear to be any essential differences between the various forms. Charteris, therefore, comes to the conclusion that it is difficult to find any justification for regarding simple hyperplasia, papillomata, fibro-adenomata or cyst-adenomata as separate entities. He regards them as varying manifestations of the one process and states that this view is strengthened by the occasional finding of transition forms between adjacent examples of these lesions.

In order to trace the views of Charteris to their logical conclusion it is necessary to follow rather closely his observations on epithelial hyperplasia. Epithelial hyperplasia of a generalized character results in the production of more or less solid sheets of cells in the smaller ducts. Sometimes only a single duct and its related acini are affected and

[1] *The Journal of Pathology and Bacteriology*, January, 1930.

sometimes changes may be found in a number of duct systems in different parts of the gland. The first change is a gradual encroachment on the lumen by small epithelial ingrowths or an irregular thickening of the cellular lining. Although these cells may be heaped up on each other, they are perfectly regular and are indistinguishable from those which ordinarily line the part. In a more advanced stage the cells become irregular in size and shape and manifest nuclear changes. Charteris points out that this appearance could not from the purely cytological standpoint be regarded as malignant, but that such cells have been seen to invade the tissues and to give rise to a tumour of similar appearance. On the other hand he found that this type of cell was more often seen in association with intraduct carcinoma. Charteris explains that when the cells become large and plump with rather indistinct outlines, the mass is almost syncytial; the nuclei are also larger and often of vesicular type with irregularities in the chromatic patterns and occasional mitoses. The cells are quite indistinguishable histologically from those which may be seen in many cancerous tumours of the breast, and yet they may be entirely confined to the ducts and acini. It is to cells of this nature that the term intraduct carcinoma is applied and they were regarded as of fundamental importance by Muir in his work on Paget's disease of the nipple (The Medical Journal of Australia, June 11, 1927). Muir shows that intraduct carcinoma may give rise either to Paget's disease by an extension up the lumen of the duct or to carcinoma of the breast by perforation of the duct wall. Charteris describes three cases in which he was able to trace the extension of intraduct carcinoma according to Muir's description until it gave rise to Paget's disease. Charteris also describes and illustrates by photomicrographs the rupture of intraduct carcinoma into the surrounding tissues and the invasion of the latter.

The general conclusion of Charteris is that the changes described by him as progressive epithelial hyperplasia are in reality only different stages of one process which finally ends in malignant disease. Under the heading of ætiology he refers to the views of Keynes which have previously been mentioned, and apparently he accepts them. In other words, chronic irritation by stimuli of a biochemical nature gives rise to the initial hyperplastic change. If the view of Charteris were accepted literally, it would mean that malignant disease would occur in every breast affected by so-called chronic mastitis, provided the patient lived long enough. It is, however, a clinical fact that chronic non-malignant changes do occur and may exist for many years without the supervention of malignant change and also that malignant disease may make its appearance without the existence of any demonstrable antecedent change comparable to so-called chronic cystic mastitis. Charteris does not contend that all breast cancer arises in the ducts, but he does hold that the ducts play a very important part in the pathological process. It is justifiable to conclude that there must be some factor other than the original stimulus to epithelial hyperplasia and that this factor brings about a disorderly and unrestrained growth among cells whose appearance at any rate is orderly, though their growth is excessive. This factor may operate at an early stage in the process of overgrowth and in these circumstances there would be, as Charteris sometimes noted, but little evidence of so-called chronic cystic mastitis with the malignant change. If the factor operated late, there would in all probability be pronounced chronic change present with the malignant manifestations. Furthermore, as already pointed out, this additional factor might not operate at all. The conception of Charteris of chronic precancerous changes as being essentially similar in spite of their varying morphological structure, papillomatous, fibro-adenomatous or cyst-adenomatous and so forth, is useful, because it indicates the likelihood of a common ætiology. His communication should be studied together with those of Muir and Lenthal Cheatle. Cheatle's work was discussed in these pages on August 18, 1923.

DIABETIC RETINITIS.

At a recent discussion before the Section of Ophthalmology of the Royal Society of Medicine P. J. Cammidge[1] reported that among one thousand patients with diabetes he had found forty-eight or 4·8% with retinitis and retinal hæmorrhages. Text books of medicine devote but little space to this question. It would naturally be presumed that the retinitis is due to the diabetes. Cammidge points out, however, that authorities are now agreed that it is doubtful whether a retinitis due to diabetes alone ever occurs. There is no relation between the severity of the diabetes and the incidence of retinitis; the existence of a definite relationship would be expected if the retinal changes were a direct result of the metabolic disturbances resulting from the primary disease. Retinitis occurs almost exclusively in patients with a mild and easily controlled glycosuria. The rapid improvement in the vision of many patients with diabetic retinitis which follows the control of the hyperglycæmia and glycosuria and the tendency of local hæmorrhages to persist suggest to Cammidge that the changes interfering with visual perception and the hæmorrhagic tendency are "to a considerable extent, at least," of independent origin. The former he regards as induced by the diabetic state and the latter by defects which are not a direct result of the metabolic disturbances induced by the diabetes. In four of his patients hæmorrhages occurred after the blood sugar had returned to normal. He shows that the hæmorrhages are associated with a lowering of the calcium content of the blood and gives clinical details in support of his statements. Diminished coagulability of the blood is an important factor in recurring hæmorrhage. There is probably a connecting link between the diabetic changes and the calcium deficiency.

[1] *Proceedings of the Royal Society of Medicine*, January, 1930.

Abstracts from Current Medical Literature.

SURGERY.

Milk Injections for Buboes.

E. Kromayer (*Klinische Wochenschrift*, December 17, 1929) advocates the use of milk injections in septic infections of the inguinal glands following soft sores. In a series of 131 patients, forty-five were treated with fomentations and only 13% of lesions resolved without further treatment, as compared with a 67% cure among eighty-six patients treated with milk. Fresh milk was heated in a water bath for thirty minutes, then passed through a sterile filter. The first injection was six to ten cubic centimetres, this was increased to ten to fifteen cubic centimetres at intervals of three to four days. Most patients required only from two to four injections. There was a definite rise of temperature with local and general reaction which soon subsided. Favourable lesions soon became painless with disappearance of fluctuation. If the injections were unsuccessful, he noted that fluctuation became more pronounced and operative interference was required earlier than if foments alone had been used. The penile lesion seemed to be favourably influenced by the injections. The period of invalidism was always greatly reduced.

The Sterility of Catgut.

Two epidemics of wound infection occurring in New York hospitals led Frank L. Meleney and Mabel Chatfield (*Surgery, Gynecology and Obstetrics*, January, 1930) to investigate the question of how the sterility of catgut can be assured. Both of these epidemics were due to the organisms of gas gangrene and six out of seven patients affected died. Cultures, made from the catgut in the same batch, in each case yielded cultures of gas gangrene anaerobes. Investigation showed the necessity for regulation in the matter and for the direction of its investigation by some recognizable authority. Information concerning catgut "accidents" is difficult to obtain and whilst the government has rigid laws concerning the sale of impure food, it is not concerned with contaminated catgut. Tests for the sterility of catgut are made by firms in some cases in a haphazard fashion. Further, these tests, unless efficient, give a false sense of security. Every single batch should be subjected to a recognized test, known to be efficient. The Hospital Standardization Committee of the American College of Surgeons promised that if the results of a study of this question were satisfactory, they would take action and recommend only the products of those firms which complied with certain rules. Eighty-three specimens of raw surgical catgut were examined and thirty-eight were found to contain pathogenic anaerobes, comprising the three common species of gas gangrene organisms. In none of thirty-eight specimens examined was *Clostridium tetani* found. In conclusion it must be assumed that any or all of the well-known gas gangrene organisms are present in catgut before sterilization and any test of sterility must be such as to bring to light any organism which may be present.

An Indication for Early Operation in Intestinal Obstruction.

William Bartlett (*Surgery, Gynecology and Obstetrics*, November, 1929) writes on indications for early operation in intestinal obstruction. The most effective non-operative treatment for paralytic ileus at present is the injection of a spinal anæsthetic. This is also applicable to many partial obstructions of the mechanical type, such as the recurring crises of carcinoma of the colon and of old intraperitoneal inflammatory disease. In the response to this treatment there is a true indication for or against enterostomy. If within fifteen minutes after the injection of the spinal anæsthetic passage of gas and fæces and disappearance of distension be not obtained, enterostomy should be performed immediately, advantage thus being taken of the anæsthesia already produced. Longer delay permits progressive depletion of salt and water reserves, interference with the circulation of the gut, absorption of toxin from the intestine and the advancement of those other processes (the nature of which is still controversial) which singly or in combination cause death. In spinal anæsthesia surgeons have a therapeutic test of value.

The Efficiency of the Surgical Mask.

Irving J. Walker (*Surgery, Gynecology and Obstetrics*, January, 1930) had his attention directed towards the question of the determination of the efficiency of the surgical mask by a series of operative infections by a hæmolytic streptococcus, which occurred at a Boston hospital in 1927. Investigation showed that three of the people associated with the operations were carriers of the hæmolytic streptococcus. The adoption of a germ-proof mask was followed by no further wound infections. In 1928 at another Boston hospital another such series of wound infections occurred and study revealed a large number of carriers of the hæmolytic streptococcus amongst the nursing and medical staff. The masks used at this hospital were found to be unsatisfactory, in that they did not cover the nose. Unsatisfactory masking, the author states, must be regarded as a weak link in the operative chain. A *questionnaire*, sent to one hundred hospitals, was replied to by sixty. In sixteen hospitals there had been seasonal epidemics of wound infection, whilst in eight it was considered that there was some relationship between wound infection and epidemics of respiratory disease. The author thinks that such a relationship exists, a view which is supported by the work of Meleney in a study of streptococcal wound infection in the Presbyterian Hospital during 1925-1926. The incidence of wound infections was found to be less in hospitals in which it was the custom to mask both the mouth and nose. Further experiments showed that masking of the mouth and nose should be carried out as a routine procedure. These experiments, the test being a severe one, were carried out on a number of masks and none was found to stand the test. A mask was then devised, consisting of two layers of gauze twenty-five centimetres (ten inches) square, stitched on three sides and between the layers of which a strip of glove rubber fifteen centimetres (six inches) square is inserted to cover the mouth and nose. This was found to be effective from the bacteriological point of view, but has certain obvious disadvantages from the point of view of comfort.

Dislocation of the Testis.

Edwin P. Alyea (*Surgery, Gynecology and Obstetrics*, November, 1929) discusses the varieties and treatment of dislocation of the testis. An ectopic testis is by no means a rare occurrence. Cryptorchidism has been found to occur in 0·1% to 0·2% of all young adults. In the vast majority it is a congenital condition due to defective development. Aberrant migration of the testis is rather uncommon. The perineal type occurs the more often and over one hundred cases have been reported. A careful survey of the literature has brought to light only twenty-three traumatic dislocations. The resultant position of the dislocated testis is dependent upon three factors: (i) anatomical abnormalities, (ii) obstruction to dislocation in particular directions, (iii) the direction and force of the blow. In one variety the testis passes through the external inguinal ring into the canal; in another group it does not pass into the canal, but lies in a subcutaneous position; lastly there is the type in which the dislocation is compound. In the subcutaneous group the new position taken up may be either pubic, superficial, inguinal, penile, perineal or crural. The ætiological factor in the majority of cases was the passage of a wheel over the genital region. Symptoms were severe shock and local pain with nausea and vomiting. Diagnosis was usually easy after the traumatic swelling had disappeared. Early diagnosis was often difficult. Treatment was very satisfactory; reduction without open operation was carried out in only three instances, open operation in sixteen. Six patients were not treated. All the patients were cured except one whose testis was in the perineal position and could not be held in the correct position; it had to be removed. There were six dislocations of the pubic type.

this being the most common. The superficial inguinal position came next with three cases; they were treated by open reduction. There were three penile dislocations, one was not treated and in the others the testis was replaced by open operation. The prognosis in all these dislocations is excellent.

Fractures of the Carpus.

HENRY H. RITTER (*Surgery, Gynecology and Obstetrics*, December, 1929) discusses fractures of the carpus. The persistent pain and limitation of function following carpal damage is distressing to the patient and annoying to the surgeon. The discomfort is not alone associated with displaced fractures and dislocations, but even with fissured fractures. The scaphoid is more often damaged than the other carpal bones. Dislocation of the semilunar is frequently associated with a fracture of either the scaphoid or articular surface of the radius. Osseous union in carpal fractures is the exception; fibrous union is the rule. Much of the disability following carpal injuries is due to intra-articular soft structure damage. Early attempts at closed reduction of scaphoid fractures or semilunar dislocations should be made. Failure to obtain proper reduction by closed manipulation should be followed by immediate open operation. Carpal fractures treated unsuccessfully by immobilization should be operated on within three months, lest irreparable subsequent arthritic changes take place. Carpal fractures due to the type of bone do not heal well, except fractures of the tubercle of the scaphoid which is definitely osseous. No fracture or dislocation of the carpus ever exists without an associated chondritis, synovitis and tenosynovitis.

Abdominal Surgery.

D. P. D. WILKIE (*The Lancet*, October 19, 1929) writes about some guiding principles in abdominal surgery. By an elaborate ritual an endeavour is made to insure that operations shall be aseptic; but ritual without reverence may be a mockery and technique associated with trauma will be tolerated less well than technique with much less perfect asepsis but gentle handling. If it were necessary to epitomize the guiding rules of the surgery of the abdomen, it might be correctly done by the phrase, "no traction, no tension." When disease or operative measures interfere with the normal relaxation of the hollow viscera, then tension, pain and trouble result. In anastomosis a single line of interrupted Lembert sutures, tied lightly so as to give the minimum of interference with blood supply, is the ideal method and the safest if the principle of detension is fully observed. In the operation of resection and anastomosis of large bowel insurance against tension by an appendicostomy or caecostomy or ileostomy, may make all the difference between a smooth and a stormy convalescence and may, indeed, be a life saving measure. Three very important elements of success are mobilization of the viscus, mobilization of its coats and drainage of its lumen. The much discussed problem as to whether drainage of the peritoneum is advantageous and effective in acute peritonitis is answered on this principle. It relieves tension and thereby improves blood supply. The fact that the drain is rapidly shut off by intestinal adhesions is undeniable, but by that time it has served its purpose. The tension in peritonitis may not prove to be due to peritonitic exudate, but to intestinal distension. It is in these cases that benefit will result from caecostomy or enterostomy, not only to relieve tension, but as an inlet for fluid to combat dehydration. In non-acute abdominal surgery, it is wise to make a complete assessment of all abdominal organs so as not to overlook other lesions. Normal findings should be recorded. In most acute abdominal conditions, when there is retention of toxic products due to some obstructive lesion, it is often very wise to plan a two-stage operation. A preliminary drainage will give the necessary relief and a respite until toxæmia has passed. Nutrition is in a measure restored, the factors of local œdema, tension, infected lymph channels and vascular stasis are eliminated and tissue calm is restored. No fixed length of time between the preliminary and radical operation can be laid down. Attention is drawn to the advantages of the Mikulicz-Paul operation of eventration of the growth in obstructing tumours of the colon. In acute intestinal obstruction it is not only the absorption of toxic products from the bowel above the obstruction that is causing danger, but other factors such as dehydration, loss of chlorides and the very important deprivation of secretions from the empty bowel below the obstruction.

Ununited Fracture of the Neck of the Femur.

F. H. ALBEE (*Surgery, Gynecology and Obstetrics*, December, 1929) discusses his results in the treatment of ununited fractures of the neck of the femur. From the standpoint of mechanical immobilization the Whitman abduction method offers everything that can be desired. If it is properly applied, it brings about ideal approximation of the fractured surfaces and provides ideal conditions for immobilization. If, however, after a trial of this method the fragments do not unite, the cause is a matter of deficient blood supply. Neither in fresh fractures nor in non-union is the use of nails justifiable. There is nothing they can accomplish that the Whitman method cannot do. In the hip there are peculiar conditions which are unfavourable to union, even in a fresh fracture, regardless of the constitutional condition or the age of the patient. These conditions are three in number. In the first place there is little or no periosteum about the head or neck of the femur. In the second place the synovial fluid which tends to flow between the fragments as soon as fracture occurs, has an inhibiting effect on callus formation. In the third place the most important is the faulty blood supply. The rate of osteogenesis is in direct relation to the flow of blood to the zone of fracture. Keith believes in the early and remarkable vascularization of bone grafts. The graft acts as a conducting scaffold for blood vessels which penetrate it from the vascular tissues overlying the trochanter and the vascular cancellous bone of the trochanter where the peg traverses it. The operation of the author consists of introducing an autogenous bone graft driven through the trochanter and neck of the femur into the loose head of the femur. The survival of the graft both as a vessel conducting scaffold and as a callus forming element depends upon complete immobilization of it in both fragments and also of each fragment with the other. Mechanically the machine-fitted peg graft fulfils this requirement in the best possible way and at the same time metabolism is being stimulated by withstanding stress because of Wolff's law.

Tumours of the Carotid Body.

A. D. BEVAN AND E. R. MCCARTHY (*Surgery, Gynecology and Obstetrics*, December, 1929) investigate and draw conclusions on the subject of tumours of the carotid body. In future neoplasms of the carotid body should not be removed when it is necessary to ligate the carotid arteries to complete the operation. If the common and the internal carotids can be saved by careful dissection, done best under local anæsthesia, the removal of a benign tumour of the carotid body will be justified. If the surgeon has definite and satisfactory evidence that the tumour is malignant, the huge 30% mortality involved in the ligation of the common carotid artery might be accepted in order to save the patient from death and from malignant disease. It is quite evident, however, that a malignant growth of the carotid gland involving these three arteries offers very little prospect of a permanent cure even by a most extensive operation. It is true of course that in a very few cases up to the present time has diagnosis been made before operation; in practically none has a definite diagnosis of tumour of the carotid body been made. The clinical diagnosis will be made more frequently in future from three points: the location of the tumour in the bifurcation of the carotid, the sense of pulsation in the tumour and the fact that the tumour is more movable from side to side and not at all from above downward and that the tumour has existed for many months or even years before the patient seeks relief.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE MEDICAL SECTION OF THE QUEENSLAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held on December 20, 1929. The meeting had been called for discussion on the undue prevalence of chronic nephritis in Queensland.

DR. R. W. CILENTO, Director, Division of Tropical Hygiene, Commonwealth Department of Health, opened the discussion. He said that the various comments made as to the failure of the Commonwealth Department of Health to institute an investigation into lead poisoning did not take into consideration the facts that there were political and commercial aspects of the problem beyond those of public health and that the Commonwealth Department of Health could not intrude into local affairs except on adequate invitation.

He sketched the literature of the subject, mentioning the conclusions reached that chronic nephritis in Queensland was the sequel of lead poisoning in the main, particularly in the twenty to forty year age group, that it was claimed at first that the source of the trouble was water and subsequently the desiccated paint of veranda railings and this owing to the fact that houses there were built on stilts and that the children were commonly kept in closed-in verandas, and when attracted to the railings to view passing traffic *et cetera*, became liberally smeared with desiccated paint which nail-biters and thumb-suckers ingested.

He then advanced various criticisms of this theory voiced by observers in Queensland and other States, namely:

1. The existence of an unexplained prevalence of nephritis in certain parts of the British Solomon Islands among the natives, where there was no paint.

2. The continued absorbtion of quantities of lead in Port Pirie among all sections of the community (as well as those engaged in the smelting works) and the absence of a definite chronic nephritis problem.

3. The fact that no adequate investigatory work had been done in Queensland, either in: (i) testing the urine of normal children from two to eight years of age in comparison with that of affected children from two to eight years; (ii) testing the dust of verandas, both the settled dust and air borne, also the lead content of desiccated paint, of washings from the hands of children who had been played on verandas and scrapings from their nails *et cetera*; (iii) testing the effect of the ingestion of carbonate of lead over a period on experimental animals; (iv) testing an adequate series of patients for a susceptibility factor, such as inherited kidney disease (familial factor), inherited syphilis (extrinsic factor), scarlet fever, rheumatic fever, streptococcal infections or other kidney-damaging exanthemata.

From a graph prepared by Dr. Cilento it was shown that from 1883 to 1916 every rise on the line of nephritis deaths above the mean level (which could be regarded as stationary and was not above that of other States) could be associated with epidemic scarlet fever, particularly if it were dissected out into the two age groups in which chronic nephritis was most obvious, namely, the group dying five years after affection with the complaint and the group dying in the twenty to forty year age period. Every epidemic in Queensland had been followed by a definite rise five years later, while the figures quoted by Dr. Croll and Dr. Nye of the twenty to forty year age group precisely corresponded with the big epidemics of 1890 and 1898-1899 in Queensland. Epidemics had occurred in 1863, 1875, 1890, 1898-1899, 1903, 1911 and 1917.

Finally it was mentioned that there appeared to be three theories which should be tested: (i) That chronic nephritis in Queensland was in the main due to lead poisoning without any other factor; (ii) that in the vast mass of children in Brisbane who ingested lead in a greater or less quantity for a long period, a certain proportion owing to some susceptibility factor showed chronic nephritis; (iii) that some other factor was responsible here for the nephritis, as it was, for example, in the Solomons, and that lead was more or less an accidental occurrence and association.

DR. D. GIFFORD CROLL congratulated Dr. Cilento on his masterly address on a very old standing complaint. He considered a full investigation into the causation of chronic nephritis was long overdue. There were numerous deaths amongst children from a cause which was unknown and which was persistent in its action. He was of the opinion that it was necessary to ascertain definitely whether there was any connexion between lead and chronic nephritis in children. Dr. Nye had made a good start with this investigation with twelve patients whom he had investigated, and who had developed chronic nephritis in later life.

Dr. Cilento referred to the absence of lead poisoning in Port Pirie and its frequent diagnosis in Queensland. Was there any evidence to show whether the Port Pirie conditions rendered lead soluble or whether it was less easily absorbed by the body?

The Western Australian statistics were of no value. There was a decline in the death rate from chronic nephritis in children and in the thirty to forty year age group in Queensland. This could be attributed to education during the last thirty years and also to legislation. There were more opportunities at the present time to take children out. They spent more time at the seaside and other holiday resorts now than thirty years before.

There appeared to be less syphilis in Queensland than in other States.

DR. J. LOCKHART GIBSON referred to the eye symptoms which he called "ocular plumbism." This was really papillœdema due to increased intracranial pressure. Patients with early ocular plumbism could be cured. There was a rapid ingestion of lead, the patient became very ill, was put to bed and thus removed from the source of the lead.

DR. JEFFERIS TURNER stated that he was not impressed with Dr. Cilento's figures or points. He would like further and fuller details of arguments of the Southern sceptics. He did not believe that their hot climate had any bearing on the increased incidence of chronic nephritis in Queensland. In his experience it did not occur in farm houses, where there were no high veranda rails and the children had more open space to play in.

Although not all cases of chronic nephritis were due to lead, they must not shut their eyes and ignore known facts, to believe in unknown and problematical causes. He had seen plenty of deaths from scarlet nephritis, which in his opinion proved Dr. Cilento's graph, but indicated nothing else. There was probably less syphilis in Queensland than in other States. There might be unknown causes; it was of no interest to them in Brisbane, if chronic nephritis occurred in the Solomon Islands where there was no bad paint.

Not all Queensland children were "leaded." He had seen many cases of only one member of the family suffering from lead poisoning, whilst the remainder were perfectly free.

DR. F. A. HOPE MICHÔD referred to the obstetric reports of Queensland and New Zealand and pointed out that the incidence of chronic nephritis was approximately eight times as great in the former as in the latter.

DR. ALEX. MURPHY considered chronic nephritis was due to a streptococcal infection. Crystalloid poisons produced interstitial nephritis and colloid poisons produced glomerular nephritis. The number of cases of chronic nephritis in Queensland was diminishing and rheumatic fever was rare. Had the campaign against tonsils anything to do with this question? Graphs showing nephritis in scarlet fever referred only to the diagnosed and not to the undiagnosed cases.

DR. W. N. ROBERTSON said that when he was on the staff of the Mater Misericordiæ Public Hospital, many nephritic patients had been referred for tonsillectomy and after removal of the septic focus the nephritic condition had

cleared up. He was of the opinion that infection had a great deal to do with chronic nephritis. Perhaps constant sweating in the tropical climates with concentration of the urine was also a cause of irritation.

DR. L. J. NYE thanked Dr. Cilento for his very excellent paper, he said it had certainly raised some very controversial points. He thought that the question of the incidence of nephritis among the black races was not quite parallel since the diseases and immunities of the blacks differed in many respects from those of the whites. Nevertheless, it all showed how necessary it was to get busy and investigate the whole question.

Dr. Nye pointed out that when he first came to Brisbane, like most other practitioners he was shocked by the extraordinary amount of chronic nephritis seen in young people. The first point in his investigations was the study of the clinical and morphological findings in this condition. These showed it to be a chronic diffuse glomerulonephritis associated with generalized vascular sclerosis, such as would be caused by some streptococcus.

Scarlet fever seemed to him to be the most probable factor, for in the light of modern knowledge it was considered that there were two varieties of nephritis, for which this *Streptococcus scarlatinæ* was responsible. The first was a non-embolic focal glomerulonephritis which occurred in the early stages of the disease. In these circumstances the damage was confined to a limited number of glomeruli. This benign type should be carefully distinguished from the second variety, a diffuse glomerulonephritis, which occurred later in the convalesence and usually manifested itself on or about the nineteenth day of the disease. It was this variety which so commonly progressed into chronic nephritis in later life. Therefore, in order to study the question of scarlet fever he had examined the records of the Brisbane General Hospital, but had been able to find no evidence that the incidence of scarlet fever nephritis was greater in Queensland than in other countries. Furthermore, he suggested that when nephritis did occur during the convalescence of scarlet fever, the signs and symptoms were surely sufficiently manifest to be brought under medical observation, but in his cases no such history was available.

As this avenue appeared closed and since the infections usually associated with nephritis were neither common nor virulent in Queensland, an effort had been made to search for some pathogenic agent which might be peculiar to that State. With this end in view eighty consecutive cases had been carefully scrutinized and inquiry into these had revealed no previous ill health in forty-two instances and in the remaining thirty-eight, fourteen patients had given a history of having had lead poisoning in childhood, while only four had given a history of scarlet fever. It was well known to all practitioners in Queensland that lead poisoning was extremely common among children there, whereas in the other States it was rarely diagnosed. He admitted that the Queensland figures were probably exaggerated and some conditions were diagnosed as plumbism without sufficient absolute proof. Such symptoms and signs as anæmia, lassitude and punctate basophilia had been considered by some medical men to be sufficient findings to warrant such a diagnosis; while it was possible that some of these conditions would not be due to plumbism, it was probable that the great majority were.

The only method by which his suspicions concerning lead poisoning could be verified was obviously by the examination of patients who had suffered from its effects some years previously. He then showed the findings in twenty such cases. This selection had been very carefully made in that only cases in which the more certain signs of lead poison was present had been included and in all either wrist drop or toe drop had been present.

These represented twenty consecutive cases of severe lead poisoning and it was seen that there was well established renal insufficiency in every instance. DR. NYE then asked how many of the patients with fewer manifestations also went on to chronic nephritis. When they saw, as had already been pointed out in a previous paper, the extraordinary number of chronic nephritics who were nail-biters, surely this suggested something more than a coincidence. He asked why they so often found in one family that only the nail-biters were affected. He quoted one such family history. A woman, aged thirty-six years, had died of goitre; she had not been a nail-biter. A woman, aged twenty-two years, had died of chronic nephritis; she had been a nail-biter. A man, aged forty-one years, was alive and well; he was not a nail-biter. A woman, aged thirty-six years, was alive and well; she was not a nail-biter. A woman, aged thirty-four years, was alive and well; she was not a nail-biter. A woman, aged thirty-two years, suffered from chronic nephritis; she was a nail-biter. A woman, aged twenty-four years, was alive and well; she was not a nail-biter. In this case it was interesting to note that the woman, aged thirty-four years, was the only member of the family to have had scarlet fever and this had happened when she was away from home.

Dr. Nye then showed another family, the children of a painter, all stunted in growth and all manifesting renal insufficiency; also two brothers, both nail-biters, of dwarfed stature with well established renal insufficiency.

Dr. Nye stated that he could go on quoting such cases for hours. It had been suggested by some physicians that the disease from which these children suffered, was not lead poisoning. He felt that this could be dismissed without any qualms. The absence of pyrexia, the typical selected areas of paralyses, the encephalopathy, the blood signs, the ocular signs, Burton's blue line, together with the environmental and circumstantial evidence left no possible opportunity for doubt in this respect.

He confessed that this conclusion to which he had been forced was entirely opposed to his original opinion, for a glomerulonephritis was usually bacteriotoxic in origin, while chemical poisons being crystalloids and of relatively low molecular weight were filtered through the glomerular tufts and were concentrated in the tubules by reabsorption of fluid from this dilute filtrate; hence they tended to produce a tubular nephritis, but the pathology in these cases could be explained by the fact that lead circulated as a colloid and as such had some selective action on the vascular endothelium, thereby causing a diffuse vascular sclerosis, as was found in these cases. He considered the whole picture was so clear cut, the association of wooden houses with dry and powdery paint, nail biting and nephritis was constant, that they must accept the fact that lead poisoning was one of the causes of chronic nephritis in childhood and that this alone was a reason for immediately instituting some much needed research.

In conclusion he said that he entirely agreed with Dr. Cilento that the question must be investigated from every point of view and that it was impossible for any individual investigator to arrive at irrefutable conclusions in the matter. Such investigation ought to be carried out by a commission. He felt that then, at least, the constant knockings of Dr. Gibson and Dr. Turner would be listened to.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected as members of the Victorian Branch of the British Medical Association:

Farmer, Paul Ward, Junior, M.B., B.S., 1928 (Univ. Melbourne), Fairfield Hospital, Fairfield.

Houghton, Percy Byron, M.B., B.S., 1928 (Univ. Melbourne), 112, Finch Street, Glen Iris.

The undermentioned has been nominated for election as a member of the New South Wales Branch:

Lee, Douglas Harry Kedgwin, M.B., 1929 (Univ. Sydney), Commonwealth Department of Health, Tropical School, University of Sydney.

LISTS OF MEMBERS.

THE lists of members of the several Branches of the British Medical Association in Australia are now available. Copies can be purchased from the office of THE MEDICAL JOURNAL OF AUSTRALIA at one shilling each.

Medical Societies.

PUBLIC MEDICAL OFFICERS' ASSOCIATION OF NEW SOUTH WALES.

THE fourth annual general meeting of the Public Medical Officers' Association of New South Wales was held in the Assembly Hall of the Education Department, Sydney, on January 28, 1930, DR. E. S. STOKES, the President, in the chair.

Annual Report.

The Honorary Secretary, Dr. H. Hastings Willis, presented the annual report for the year 1929 and commented upon it section by section. He moved its adoption. The report is as follows:

The year 1929 has been a relatively quiet one for the Public Medical Officers' Association of New South Wales, the political situation, both Federal and State, having been unfavourable for the prosecution of many of our schemes. Nevertheless a careful watch has been kept over members' interests and appropriate action taken when necessary.

The Committee met thirteen times during the year, the attendances of members being as follows:

Dr. E. S. Stokes (President)	12
Dr. M. R. Finlayson	6
Dr. S. Evan Jones	9
Dr. A. E. Machin	10
Dr. Dorothy McClemens	10
Dr. E. L. Morgan	10
Dr. H. H. Nowland	5
Dr. J. S. Purdy	6
Dr. C. J. M. Walters	2
Dr. H. M. Taylor	5
Dr. H. Hastings Willis	13

During Dr. Finlayson's absence of six months from Australia Dr. Taylor acted for him.

The membership of the Public Medical Officers' Association advanced during the year to 86 and at the end of the year had receded to 79 due to retirements and resignations from the services. Members are asked to persuade junior medical officers to join in larger numbers.

The organization of associations of public medical officers in other States proceeds slowly. As in former years the policy of urging the formation of independent organizations rather than branches of this association has been followed. A suggestion has been made that where no organization exists in a State, members from that State might be accepted as associate members of an association in another State. This association has made no provision for associate members in its rules.

A conference of public medical officers from the Australian States was held in Sydney on September 2, 1929. Study leave and the organization of public medical officers' associations in the various States were the subjects discussed.

Our relations with other professional and service organizations remain cordial and unchanged.

The Council of the New South Wales Branch of the British Medical Association has again refused our repeated request for direct representation on the Branch Council.

Circular letters distributed to members during the year numbered four.

The annual social function took the form of a motor excursion and luncheon at Bulli Pass and Cataract Dam on Saturday, September 7, 1929, when members were pleased to be able to extend hospitality to Dame Janet Campbell, of the British Ministry of Health, and public medical officers attending the Australasian Medical Congress from other States. The party numbered seventy-nine.

Facilities for post-graduate study have not been increased during the year. The New South Wales Branch of the British Medical Association has recently become interested in this matter and the results of its labours are awaited with interest. Much is also expected of the new Sydney School of Hygiene and Tropical Medicine which is shortly to function.

During the year an effort has been made to secure a minimum salary of £750 *per annum* for medical officers employed on construction works. The salaries of most of our members have been controlled during the year by the association's agreement with the New South Wales Public Service Board which expires on June 30 next. Some agitation was necessary early in the year to secure payment of certain moneys due under this agreement.

In pursuance of our policy against the lay control of medical activities the New South Wales Government was asked to consider the appointment of a medical officer from within the services to the position of chairman of the Hospitals Commission. A layman from another State was appointed.

Support has been accorded the proposals of the New South Wales Public Service Association for amendment of the New South Wales *Superannuation Act*.

It has been noted with regret that on occasions senior medical positions under the New South Wales Government are filled from without the services.

Early in the year representations were made to the New South Wales Government and Public Service Board against a tendency to practise economy at the expense of efficiency by leaving an unduly large number of medical positions unfilled, especially in the mental hospitals. Towards the end of the year this practice was less prevalent.

From time to time during the last two years individual members have stated that the travelling and subsistence allowances made by the New South Wales Government were inadequate. After consideration the committee feels that there is a case for action and is awaiting detailed statements of actual receipts and expenditures before addressing the appropriate authority on the matter.

It is doubtful whether the opportunities for promotion in the services are as good as our members have a right to expect. In this connexion the Public Service Board of New South Wales has been asked to consider the interests of members in filling the vacant position of Principal Medical Officer in the New South Wales Education Department and has been informed of the Committee's opinion that the creation of an impression that the higher positions in the New South Wales medical services are closed to those who as juniors seek careers in those services, is inimical to recruitment from the best type of graduates.

A financial statement for the year is appended. It will be noted that continued good fortune and the practice of economy have resulted in an appreciable addition to the reserve fund, but the committee thinks that the time has not yet come for any reduction in the amount of the annual subscription.

DR. A. A. PALMER seconded the motion and congratulated the committee on its activity during the past year. He objected, however, to the paragraph concerning direct representation on the Branch Council of the British Medical Association. He said that it was not fair to blame the Council for not doing what it had not the power to do. It was of no use making repeated requests to the Council on this matter. An alteration of the constitution would be necessary and if the Public Medical Officers' Association wished to pursue the matter further, it would have to take steps to have that constitution altered.

The Secretary in reply said that he agreed with Dr. Palmer that little would be gained by again approaching the Council under present conditions. He doubted, however, whether the matter was of such importance as to warrant the committee undertaking a troublesome and unpleasant agitation for reform of the constitution of the New South Wales Branch of the British Medical Association. Moreover, he thought that success of such an enterprise would be doubtful unless the Branch Council gave its support. As he understood the Council was unwilling to give this support, he thought it not unfair to blame the Council for the present state of affairs.

The report was adopted.

Election of Office-Bearers.

The election of office-bearers for the ensuing year resulted as follows:

President: Dr. J. A. L. Wallace.
Honorary Secretary: Dr. H. Hastings Willis.
Honorary Auditor: Dr. Elsie J. Dalyell.
Members of the Committee: Dr. H. W. Palmer, Dr. J. S. Purdy, Dr. E. L. Morgan, Dr. S. Evan Jones, Dr. H. H. Nowland, Dr. Dorothy McClemens, Dr. M. R. Finlayson and Dr. H. H. Willis.

Financial Statement.

DR. E. L. MORGAN, Honorary Treasurer, presented the financial statement for the year. It showed a credit balance of £200 10s. 9d., including a reserve fund of £154 16s. 8d.

DR. J. A. L. WALLACE, Honorary Auditor, in reporting on his audit of the books said that as in previous years he was agreeably surprised at the great care Dr. Morgan had taken to record even the minutest detail of expenditure. He had also been very diligent in the collection of subscriptions in arrear. He thought the Association was very fortunate in having such a treasurer and he hoped he would be willing to serve for the ensuing year.

DR. E. SYDNEY MORRIS seconded the adoption of the statement which was carried.

DR. S. EVAN JONES moved that the annual subscription of temporary and probationary medical officers during their first year in the services be ten shillings instead of one guinea. He pointed out that this class of medical officer had not joined the Association in satisfactory numbers and he thought a reduced subscription for one year would effect an improvement. The motion lapsed for want of a seconder, Dr. Evan Jones remarking that this negative expression of opinion would be interpreted as support of the committee's recommendation that the subscription be not reduced.

Publicity.

DR. D. S. FRASER suggested that the time had come for the issue by the Association of a quarterly journal to include the news now published in the circular letters together with matter of medical and scientific interest. He thought the Association had not indulged in enough publicity.

The secretary asked that no motion be carried, but that the suggestion be left for the committee to consider. The question of publicity had been considered from time to time, but the issue of the circular letters had been the only tangible result. He reminded the meeting that the Federal Health Department's journal, *Health*, had had more chances of success than would the journal now suggested in that it was financed by the Federal Government and edited by a medical officer as part of his departmental duties, yet that journal had died of inanition. It was by no means easy to secure a supply of suitable matter for such a journal. However, he would explore the matter further and would bring it before the new committee.

The secretary reported that the committee had no proposal yet to make on the suggestion of the last annual meeting that a prize for preventive and psychological medicine be founded at Sydney University. The matter was not urgent and would be further considered. It was doubtful whether the present financial position of the Association was strong enough to permit of this expenditure.

Defence of Members.

He reported that the committee had also no proposal to offer concerning means of defending members who found themselves in trouble as a result of their duties as public medical officers. He directed attention, however, to the special meeting of the New South Wales Medical Union called for January 23, 1930.

DR. A. A. PALMER, at the request of the president explained these proposals of the Medical Union's Council.

The Honorary Treasurer, Dr. Morgan, said that his interpretation of the rules would not permit him to spend any of the Association's funds in helping a member in trouble. He invited anyone who wished the rules altered, to submit an appropriate motion. At present only moral support could be given by the Association.

DR. A. A. PALMER thought it absurd for an association which was not financially strong enough to found a £5 prize, to think for a moment of going into such an expensive and risky business as medical insurance. All members should belong to the Medical Union which would help them if they had a just cause, as also would, he felt sure, the governments they served. Public medical officers were in a better position in that respect than were private practitioners, he thought. In reply to an interjection questioning the necessity of joining the Medical Union he said he would no more think of practising his profession either as a government servant or as a private practitioner without membership of the Union than he would think of driving a motor car without insurance against third party risk. He would not be able to sleep at night unless he was covered in that way.

DR. FINLAYSON and DR. EVAN JONES also spoke, but no motion was submitted.

The meeting concluded with a hearty vote of thanks to the retiring President, Dr. E. S. Stokes, for his services during the year, carried by acclamation on the motion of Dr. J. A. L. Wallace.

Correspondence.

THE ÆTIOLOGY OF RODENT ULCER.

SIR: In reply to Dr. Molesworth's second letter, I would like to try and unravel some threads from this tangled skein, in which mis-statements, hair-splitting and jockeying with words seem to play the most prominent part. However, before doing this, it seemed to me that some further references on rodent ulcer due to trauma would not be out of place. In recording references I have tried to keep to English and American works for the following reason, which I extract from an article by Dr. Adamson (*The Lancet*, March 21, 1914), in which he says: "Rodent ulcer, ever since it was first described by Jacob, of Dublin, in 1827, has continued to attract great attention in this country. It has been looked upon by most British observers as distinct in its clinical features and its pathology from other forms of malignant epitheliomata of the skin. Continental authorities, with but few exceptions, which include particularly Unna, of Hamburg, and Dubreuilh, of Bordeaux, have not until recent years recognised rodent ulcer as a special type of epithelioma." (Dubreuilh was the only Continental authority I quoted in my last letter.) I now quote from the well-known text book of R. MacKenna, of England ("Diseases of the Skin," 1927). Under the ætiology of rodent ulcer he states: "The present cause is unknown, but chronic irritation and injury are undoubtedly predisposing causes." Ormsby, of America, in his book ("Diseases of the Skin," 1927) states: "The essential cause of carcinoma is unknown; many factors enter as predisposing causes, among these trauma, both mechanical and chemical, is important." (The ætiology of rodent ulcer is considered with carcinoma of the skin.) In THE MEDICAL JOURNAL OF AUSTRALIA, March 1, 1930, Professor Welsh, in an article "On the Nature, the Origin and the Spread of Cancer and Tumour Growth" *et cetera*, states: "On the Origin and Genesis of Cancer and Tumour Growth. . . . Already we know some of the predisposing causes of cancer and tumour growth in local irritation and trauma, in the influence of heredity and senescence, in the developmental inclusions and embryonic residues of Cohnheim." I will now quote from Darier, one of Dr. Molesworth's authorities. It was published in 1920 and is an authorized translation by S. Pollitzer. In this, under ætiology and pathogenesis of the epitheliomata, he says: "The cause of the facial localisation of the vast majority of epitheliomata of the skin is not known; undoubtedly it is to a considerable extent because this region is the seat of election of senile degeneration and leukoplakia; because it is particularly exposed to traumatisms, to inoculation of infectious germs, to the influence of atmospheric factors and especially of light, which seems to exert a favoring

action, and possibly because the complicated planes of embryonic growth in the face afford a ready opportunity for the misplacement of epithelial cells."

In regard to what I regard as the least important cause of all, namely, their origin from moles, I quote from Sir Norman Walker's latest text book, 1924, in which he states: "I believe, however, that Sir Benjamin Brodie was correct when he drew attention to its frequent origin from moles."

At a meeting of the New South Wales Branch of the British Association of Dermatology held in the Maitland Lecture Hall at the Sydney Hospital on March 13, 1930, I showed two further cases of rodent ulcer due to injury. At that meeting Dr. Langloh Johnston, Dr. Hamilton, Dr. Dawson, Dr. Lindeman, Dr. Raffan and Dr. J. Witton Flynn, the only speakers, all expressed definitely and emphatically their views that rodent ulcer could be and was produced by trauma, at the same time instancing cases which they had seen and treated. Moreover, they were in entire agreement with the diagnosis of the two cases which I showed, as well as the one due to a scratch from a pin on the forehead which I had exhibited previously and again showed by a lantern slide from the photograph.

Now for a consideration of Dr. Molesworth's letter. He said in reference to himself: "I may have expressed grave doubts as to whether trauma could play the part of an originating cause." Now there is no "may have." He either did or he did not and this he adds after challenging the veracity of my statement on this subject. Surely this is irreconcilable with the following statement in his first letter: "Failing in these, logic demands that he abandon trauma as an explanation except in rare cases of rodent ulcer." His next statement is: "In any case, why bring up a 1905 edition (Crocker) or even a 1920 edition (MacLeod)? Stelwagon's edition was like the rest, not quoted, but was, I think, 1910." Stelwagon's edition was in fact 1918. I make no apology for quoting these authorities (Crocker and Stelwagon), even as I stated to show that "the view that trauma is a causative factor in rodent ulcer is not a new one," because in dealing with rodent ulcer we are not dealing with a new disease, as it was first accurately described by Jacob, of Dublin, one hundred and three years ago; because their works represent the two greatest masterpieces in English and American dermatological literature today; because they both stated definitely that rodent ulcer was due to trauma. Radcliffe Crocker stating: "A certain number date from a local injury" and Stelwagon stating: "The disease has often been noted to start at the site of a scratch, cut or other accidental traumatism." There is only one meaning to these statements, except to anyone trying to avoid the issue. I do not know whether Dr. Molesworth purposely avoided the word "often" in quoting Stelwagon's description, but this he did. Probably this will make the statement more definite to him.

In reference to his statement: "In any case why bring up . . . . even a 1920 edition (MacLeod)?" Now I would refer my colleague to an article of his written in 1927 on rodent ulcer in which he prefers not to record references (his exact statement will be noted later) and yet he makes seven references and out of these seven references, recorded on his article on rodent ulcer, 1927, four are from MacLeod's book. Surely he ought to accept one reference by myself, from the same book and author on the same subject. The only difference is that my reference is damning to his cause. Of the seven references above referred to, two are from Darier, "*Précis de Dermatologie*," 1909. This book of Darier's was written only five years after Crocker's and is nine years older than Stelwagon's, yet Dr. Molesworth states: "One might almost as well quote Aristotle or Burton's 'Anatomy of Melancholy'." What has he to say about this? Finally, in regard to the dates of authorities, it is not the date, especially in regard to a malady described one hundred and three years ago, but the scientific value of the article or statement which is of importance. As an instance of this I quote some references with dates on cancer of the skin, from Dr. Sequeira's latest text book ("Diseases of the Skin," 1927). These references include amongst others Norman Paul, "Influence of Sunlight," H. K. Lewis, 1918, and amongst the earlier references Adamson, *Lancet*, 1908; A. Bowlby, 1894; S. Nicolau, 1905; J. H. Sequeira, 1901. I am sure Dr. Sequeira would be pleased to learn, according to Dr. Molesworth, that "he might almost as well quote Aristotle or Burton's 'Anatomy of Melancholy'" as the above earlier references.

Again, Dr. Molesworth states: "And it is a fair legal burden that if you quote authority on one point, you have to accept him on all. Dr. Paul knows that neither Crocker nor even MacLeod so much as mention the influence of sunlight as a possible cause of rodent ulcer." I grant this legal burden on all subjects discussed by an author, but not on subjects not discussed. Moreover, I know that it is not accepted by the majority in England and the Continent that rodent ulcer is due to sunlight, even today.

Now for Dr. Molesworth's authorities. They mostly seem to support trauma as a causative factor. Darier did, as quoted above. Again, in reference to Darier, Dr. Molesworth states: "That he is aware of the rôle of sunlight as shown by the following taken from page 471 (1928 edition): 'In the case of persons exposed to the inclemencies of weather, sailors, motorists, aviators, coachmen, farmers, and mountaineers, there are to be observed on the uncovered parts of the skin changes analogous to those of xeroderma (? *pigmentosum*) and of senile degeneration . . . . all by a skin affected with chronic radiodermatitis to the appearances produced by xeroderma and presenile dystrophies. The analogy extends to the epitheliomatous complications which are equally frequent'." In regard to this statement, is Dr. Molesworth aware that in *xerodermia pigmentosa*, prickle cell carcinoma and not rodent ulcer is the accompanying malignant growth? So that this statement like many others is irrelevant to the subject under discussion. Any opinions of Jadassohn and Whitfield not being published, I pass by, as this is not a romance on dermatology; although in regard to the former Dr. Molesworth states: "That he would deny the possibility I gravely doubt." Apparently he is a believer in the traumatic theory, so that Dr. Molesworth's authorities mainly support my own observations on thousands of cases of rodent ulcer which I have seen and treated.

On the question of sunlight in the production of rodent ulcer, it was only years after I had promulgated the sunlight theory here, that Dr. Molesworth accepted and propounded it with such wild enthusiasm that today he refers to it as his hypothesis. This is what Dr. Sequeira says in his text book ("Diseases of the Skin," 1927): "Norman Paul points out the frequency of cutaneous carcinoma in Australia and lays stress on the irritative effects of actinic light." In July, 1917, I wrote, and there appeared in my book published in 1918, the following *inter alia*: "The most important cutaneous lesions viewed from the standpoint of their prevalency and destructive effects, are those malignant diseases which are especially prone to occur on exposed parts, foremost amongst which are rodent ulcer and epithelioma. Factors which play a significant rôle in their causation and prevention are the actinic rays of light, and the pigmentation of the skin. If we follow the geographical distribution of the coloured races, in whom the pigmentation of the skin is most pronounced, we find that they occupy tropical and subtropical countries, in which, although the effects of sunlight are most severe and long continued, its action upon them is minimal. This is verified by the opinion of authorities on tropical diseases, and Castellani states that these diseases are rare in the natives of Ceylon. It will, therefore, be seen that the melanin, the pigment of the skin, stands as a sentinel guarding the underlying tissues from the baneful effects of sunlight. In the white races this pigment is confined for the most part to the periphery of the cells of the basal layer of the epidermis and to the lowermost stratum of the prickle cell layer, whilst the pigment granules may be found in the interepithelial lymphatic spaces and in the fusiform connective tissue cells of the papillary body. In the dark races, MacLeod states that pigment is to be found as high up as the transitional layers of the epidermis, as well as in some of the connective tissue cells of the superficial portions of the

corium. But the white races have the power of reacting to the external stimulus of light, with an increase in the amount of pigmentation. This phenomenon is to be seen in those who are exposed to strong sunlight when unaccustomed to it. Almost immediately, the subjacent tissues become swollen and painful and the skin assumes a bright red colour followed by desquamation (*erythema solare*). Later a more permanent brownish pigmentation is produced which has the property of excluding the actinic or ultra-violet rays of the spectrum. The above-described results are to be attributed to these rays and not to the effects of heat" *et cetera.*

As will be seen, all this writing about sunlight proffered by Dr. Molesworth to one who was a pioneer of this expression of opinion, is suggestive of the case of the student wishing to teach his master the subject which he has taught him. When writing on this subject ten years later, Dr. Molesworth states: "In conclusion I wish to point out that though in this work I have only rarely mentioned authority, the reason for this is not that I neglected or despised authority, but that little or no useful purpose would be served by discussing the different views which have been expressed, and that it would be better to start *de novo.*" As his was supposed to be an original article for an M.D. thesis, it will be seen from the above extract from my book, it was an unfortunate omission to avoid references, especially Australian references, and more especially in view of the fact that the pathological aspect in Australia had been fully investigated by Professor Cleland and myself (THE MEDICAL JOURNAL OF AUSTRALIA, May 1, 1920) and later by myself (THE MEDICAL JOURNAL OF AUSTRALIA, January 23, 1923). However, two references in his article, as I have stated, were made to Darier (1909) and four to MacLeod (1920).

Although I have always recognized the importance of embryonic residues in dermatological practice and in regard to rodent ulcer, the longer I practice dermatology the more convinced and the greater importance I attach to these embryonic residues. Those multiple basal cell epitheliomata, especially of a superficial type, which have so frequently been noted and described of recent years and which occur on covered parts of the body, must be ascribed to these embryonic residues. They certainly cannot be due to light or trauma. It is also my opinion that on the face the actinic rays of light might stimulate these latent embryonic residues to activity.

In regard to basal cell epitheliomata due to trauma and those originating beneath keratoses, I have drawn attention to certain differences in their structure to three types of basal cell epitheliomata of which I described the pathological features under the headings of the reticular type, the budding type and a combined budding and reticular type (THE MEDICAL JOURNAL OF AUSTRALIA, January 27, 1923). The three latter types I regard as representing growths whose origin lies in the pilo-sebaceous apparatus or whose ultimate futile aim was the development from the basal cell layer of pilo-sebaceous follicles. On the other hand a fourth type represented by basal cell epitheliomata due to trauma and those originating beneath keratoses, represents not a development of the pilo-sebaceous apparatus, but merely a proliferation of the epidermis in which the fibrous tissue may play an important part in the moulding.

In conclusion I would like to express my surprise at Dr. Molesworth's assertion that certain growths, "for all we can tell, they, like Topsy, just growed." This, surely, is a worthy contribution to a scientific subject.

Yours, etc.,

NORMAN PAUL.

"Harley,"
143, Macquarie Street,
Sydney.
March 15, 1930.

LEAD POISONING IN QUEENSLAND.

SIR: Your sympathetic leader in the issue of March 8 is gratifying to those of us in Queensland who have been fighting for preventive medicine for so many years. May I correct a misconception in your leader. Our legislature has not been so apathetic as you think. We succeeded "off our own bat" in having a law passed, which came into operation in 1923, making it penal to apply paint containing lead to surfaces within the reach of young children. We specially emphasized veranda railings and garden fences. We did not ask to have lead paint abolished from other surfaces. We have reason to believe that this law has been often disregarded by master painters. A salutary lesson occurred a year or two ago, when a prominent master painter was compelled, in order to avoid prosecution, to remove all the lead paint from a finished job and to replace it with zinc paint at his own cost. The knowledge of the danger of lead paint has been gradually acquired by parents, but they are at the mercy of master painters and Mr. Editor, "zinc paint lasts too long." It also "lasts longest when no lead is mixed with it." These quotations are both from the mouths of master painters. I have proof of the truth of the last quotation.

Yours, etc.,

J. LOCKHART GIBSON, M.D. (Edin.).

Brisbane,
March 14, 1930.

Post-Graduate Work.

POST-GRADUATE COURSE IN SYDNEY.

IT has been arranged to hold a post-graduate course of study in Sydney from Monday, May 26, to Friday, June 6, 1930. The course will be under the direction of the New South Wales Branch of the British Medical Association. The course will be a full time course and will occupy the whole of each day with the exception of Monday, June 2, which is a public holiday. The programme has been arranged to suit the requirements of general practitioners and will be almost entirely clinical. The course will include medical and surgical demonstrations. These will be held concurrently and those taking the course will have the option of attending demonstrations *et cetera* at any given time on either a medical or a surgical subject. Demonstrations will also be held in diseases of children, obstetrics, gynæcology and special subjects. The detailed programme will be published at an early date. The fee for the course will be four guineas.

ANNUAL COURSE IN BRISBANE.

THE Council of the Queensland Branch of the British Medical Association announces that it will hold its annual post-graduate course in Brisbane from June 2 to 6, 1930.

Dr. S. V. Sewell, of Melbourne, will deliver a lecture on medical subjects and will give a demonstration. Dr. Alan Newton, of Melbourne, will deliver a lecture on the surgery of the upper regions of the abdomen and will demonstrate some abdominal operations. Dr. R. Gordon Craig, of Sydney, will deliver the Bancroft Oration on June 6, 1930. The title of the oration will be "Progress in Prostatic Surgery." It will be illustrated by a cinematographic film prepared by the American College of Surgeons on prostatic enlargement. Dr. G. A. C. Douglas, of Brisbane, will give a demonstration on the treatment of fractures of the femur.

The subscription to the course is two guineas. This will include attendance at the lectures and demonstrations and also a double ticket for a dance and bridge party to be held on June 2, 1930.

Members are asked to notify the joint Honorary Secretaries, Dr. Neville G. Sutton and Dr. Keith Ross, British Medical Association Building, 35, Adelaide Street, Brisbane, as early as possible, that they intend to be present and to intimate whether or not they will be accompanied by their wives.

Books Received.

RESEARCH AND MEDICAL PROGRESS AND OTHER ADDRESSES, by J. Shelton Horsley, M.D.; 1929. St. Louis: The C. V. Mosby Company; Melbourne: Stirling and Company. Post 8vo., pp. 208. Price: $2.00 net.

HYPERTENSION AND NEPHRITIS, by Arthur M. Fishberg, M.D., 1930. Philadelphia: Lea and Febiger. Royal 8vo., pp. 582, illustrated with 33 engravings and one coloured plate. Price: $6.50.

BIBLIOGRAPHY WITH SYNOPSIS OF THE ORIGINAL PAPERS OF THE WRITINGS OF SIR JAMES MACKENZIE, M.D., F.R.C.P., LL.D., F.R.S. Edited by W. B. R. Monteith; 1930. Humphrey Milford, Oxford University Press. Demy 8vo., pp. 100.

HEALTH WITHOUT FADS, A COMMON-SENSE MANUAL, by Robertson Wallace, M.B., C.M., 1930. London: Methuen & Company Limited. Crown 8vo., pp. 120. Price: 3s. 6d.

DEMONSTRATIONS OF PHYSICAL SIGNS IN CLINICAL SURGERY, by Hamilton Bailey, F.R.C.S. (Eng.); second edition, revised and enlarged; 1930. Bristol: John Wright and Sons Limited. London: Simpkin Marshall Limited. Royal 8vo., pp. 279, with 306 illustrations, some of which are in colour. Price: 21s.

AFFECTIONS OF THE EYE IN GENERAL PRACTICE, by R. Lindsay Rea, B.Sc., M.D., M.Ch., F.R.C.S.; 1930. London: H. K. Lewis and Company Limited. Demy 8vo., pp. 163, with seven coloured plates and thirty-three other illustrations. Price: 10s. 6d. net.

A SHORTER SURGERY: A PRACTICAL MANUAL FOR SENIOR STUDENTS, by R. J. McNeill Love, M.B., M.S. (Lond.), F.R.C.S. (Eng.); Second Edition; 1930. London: H. K. Lewis and Company Limited. Demy 8vo., with seventy-four illustrations including thirty-one plates (one coloured). Price: 16s. net.

Diary for the Month.

APRIL 1.—New South Wales Branch, B.M.A.: Council, Election of Standing Committees.
APRIL 2.—Victorian Branch, B.M.A.: Branch.
APRIL 3.—South Australian Branch, B.M.A.: Council.
APRIL 4.—Queensland Branch, B.M.A.: Branch.
APRIL 8.—New South Wales Branch, B.M.A.: Organisation and Science Committee.
APRIL 8.—New South Wales Branch, B.M.A.: Ethics Committee.
APRIL 8.—New South Wales Branch, B.M.A.: Post-Graduate Work Committee.
APRIL 10.—New South Wales Branch, B.M.A.: Clinical Meeting.
APRIL 10.—Victorian Branch, B.M.A.: Council.
APRIL 11.—Queensland Branch, B.M.A.: Council.

Medical Appointments.

Dr. Edward James Hamp, M.R.C.S., L.R.C.P., has been appointed Resident Medical Officer at the Adelaide Hospital, Adelaide, South Australia.

* * *

Dr. H. L. Chester (B.M.A.) has been appointed Medical Officer of Health by the Koora Road Board, Western Australia.

* * *

Dr. Malcolm Bell (B.M.A.) has been appointed Medical Officer of Health, Bruce Rock Road Board, Western Australia.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xvi.

SYDNEY HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Vacancies.

ROYAL HOSPITAL FOR WOMEN, PADDINGTON, NEW SOUTH WALES: Honorary Assistant Surgeon.

THE BRISBANE AND SOUTH COAST HOSPITALS BOARD: Honorary Vacancies.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 21, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting apopintments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Mount Isa Hospital.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Medical practitioners are requested not to apply for appointments to positions at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

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